Dear Rt Hon Jacinda Ardern, Prime Minister, Hon Andrew Little, Minister of Health, Hon Dr. Ayesha Verrall, Minister of COVID-19 Response, and Hon Peeni Henare and Hon Aupito William Sio, Associate Ministers of Health

In this Open Letter and evidentiary document, I share my research results on overseas government and Ministry of Health (MoH) COVID-19 vaccine surveillance and pharmacovigilance data indicating irreparable vaccine-induced harm. Furthermore, I share important evidence that SARS-CoV-2 originated from gain-of-function research, remind you that no evidence exists for an animal-to-human origin, and highlight that its potential source lay beyond Wuhan, China. A series of requests for investigations are made below linked to this evidence, including the statistical *biases* evident in the Ministry of Health and other healthcare agencies' calculable unvaccinated COVID-19 case rates. These biases essentially eliminated the negative vaccine effectiveness harm signal from ready public view. This evidentiary document is provided by a former European corporate venture capital-funded CEO/vaccine innovator ("Vaccines for Mutating Viruses"), a veterinarian with 36 years of vaccine use experience, and a private researcher. It is supported by 525 unique data, scientific, and other citations.

According to New Zealand, England, Scotland, and Canada healthcare agencies and Global surveillance data (77 nations), these vaccines failed to prevent SARS-CoV-2 infection as initially touted. Significant negative vaccine effectiveness and vaccine failure were evident with the emergence of antigenically distinct strains (i.e., Delta, Omicron). The vaccine industry experienced antibody-dependent enhancement of virus infection (ADE) and vaccine-associated enhanced disease (VAED) with three different coronaviruses and their spike protein vaccine prototypes in the last 30 years, giving my study results a predictable context. Furthermore, one year of US lot-numbered COVID-19 vaccine-associated deaths and hospitalizations equaled 32x (Comirnaty 15.4x) and 20x (Comirnaty 10.5x) of all US vaccine-associated deaths and hospitalizations, respectively. These adverse outcomes were highly skewed and peaked across vaccine lots and were associated with a minority of lots sent to a larger number of US States. This data highlights that there was an urgent need for investigation by the US and other regulatory and healthcare agencies before expanded population use.

A vast chasm exists between the vaccine safety and efficacy experienced in 2021-2022 and the falsifiable 95% vaccine efficacy and safety proclaimed by governments with Comirnaty's first Emergency Use Authorization in 2020 (USA). This document reviews critical pharmacotoxicology and clinical safety package deficiencies evident in overseas regulatory reviews. This helps explain why Pfizer then struggled to cope with the sheer volume of Comirnaty adverse event reports in the first 90 days post-launch. This was uncharacteristic of a safe vaccine. Numerous vaccine-associated enhanced disease mechanisms are evident by which vaccine spike proteins can cause disease or exacerbate comorbidities common to severe COVID-

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19 outcomes. These mechanisms place upregulated furin and angiotensin-converting enzyme-2 receptors (ACE2) and prevalent comorbidities in tissues and organs common to all three center-stage. At the same time, SARS-CoV-2's spike protein provides its uniquely encoded furin cleavage site for the furin to cleave its S1 and S2 sub-units and activate its ACE2-receptor-mediated infectivity and pathogenicity.

Of grave concern for global public health is a gain-of-function origin to SARS-CoV-2 is indicated by its spike protein incorporating human infectivity and pathogenicity enhancing features unprecedented in nature while synthetic biology left its fingerprints. Furthermore, there is no evidence supporting a Wuhan Huanan market zoonosis because no virus progenitor or animal host was ever identified. There are two reasons for detailing a coronavirus gain-of-function origin to SARS-CoV-2. Firstly, the negative vaccine effectiveness evident in governments' COVID-19 surveillance data could have been enhanced by a genetically modified SARS-CoV-2. Secondly, the world will be left vulnerable to future pandemics if there was no accidental release from the Wuhan Institute of Virology. At least two other potential SARS-CoV-2 origins exist beyond Wuhan, with one of these potentially involving a WHO, Five Eyes, and NATO-spearhead member nation connected with Ukraine.

The US Department of Defense (DoD) and National Institutes of Health (NIH) funding of EcoHealth Alliance (EHA, $69 million) and its connections one-degree-removed were scrutinized because EHA's leader led a failed attempt to *cover up* SARS-CoV-2's gain-of-function origin. EHA directed research that genetically modified bat SARSr-CoVs that could not infect humans so that they could. EHA's $14.2 million funding application to the DoD in 2018 showed its intent to insert a codon-optimized furin cleavage site (FCS) into bat SARSr-CoVs. A uniquely encoded Arginine-doublet containing FCS now sits between SARS-CoV-2's spike protein S1 and S2 sub-units, which has no precedent in known viruses and may have infringed patents. Besides EHA's long-standing collaborations with two coronavirus gain-of-function research epicenters in the USA and China, it had another with Metabiota. Metabiota's Series-A lead investor was a Hunter Biden part-owned investment firm. The DoD-funded Metabiota operated in Pentagon Biolabs in Ukraine and US-funded Biolabs in Cameroon and researched corona-, monkeypox-, influenza-, and Ebola viruses. Metabiota has implemented major DoD and Homeland Security contracts across Central Africa while its surveillance role in Sierra Leone's Ebola outbreak in 2014 created significant controversies.

You are requested to investigate: (1) this New Zealand and overseas evidence for negative vaccine effectiveness, vaccine failure, and toxic vaccine lots, (2) the statistical biases evident in the MoH and other healthcare agencies' calculable unvaccinated COVID-19 case rates, which essentially eliminated the negative vaccine effectiveness signal, (3) the role of COVID-19 vaccination in *exacerbating* comorbidities most frequently associated with serious-severe COVID-19 outcomes, (4) SARS-CoV-2's gain-of-function origin while internationally championing a punitive global ban on gain-of-function R&D, and (5) the

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conduct of the WHO during COVID-19 linked to seven critical points detailed in section 2.7. Would you please ensure New Zealanders are updated on their recently acquired life-long health risks and that informed consent guidelines associated with COVID-19 vaccination be urgently amended? Would government please prioritize clinical research into COVID-19 antibody-dependent enhancement of virus infection, vaccine-associated enhanced disease, and antigenic imprinting in the New Zealand population?

Thank you.

Yours sincerely

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1 COVID-19 VACCINE INEFFECTIVENESS, HARM & TOXICITY

Part 1 Organization: The evidence for COVID-19 vaccine harm is available via numerous private research studies: **(1)** Our World in Data (OWID), and New Zealand, England, Scotland, and Canada results (**hyperlink** to detailed results and annotated graphics),[1] and **(2)** Vaccine Adverse Event Reporting System (VAERS) toxic COVID-19 vaccine lot results (**hyperlink** to detailed results and annotated graphics)[2] (via my website blogs). If these results are no longer available online, please request a copy by email (covid19vaccinesafetynz@protonmail.com). Sections 1.1.1 – 1.1.4 represent the global and national evidence for vaccine-associated enhanced SARS-CoV-2 infection, hospitalization, and death rates. Section 1.2 presents an analysis of one year of vaccine adverse events using data from the US Center for Disease Control (CDC) Vaccine Adverse Event Reporting System (VAERS).

The enhanced rates of COVID-19 infection and disease on evidence associated with COVID-19 vaccination are then biologically explained in sections 1.1.5-9, while the results for the VAERS toxic-harmful vaccine lots are explained in sections 1.2.1-2 and 1.3. Reviewing this population-level vaccine effectiveness and safety-mortality data in 2022 behooves us to explain the **chasm of difference** between what has arisen in 2021/2022 versus the claimed 95% efficacy and safety narrative touted with the first Emergency Use Authorization (EUA) of COVID-19 vaccines in December 2021. The aim of sections 1.4 and 1.5 is to explain from a vaccine development perspective how this great chasm of difference arose.

As a general comment applicable to sections 1.2.1-1.5.4, Comirnaty's safety and efficacy were **prioritized for scrutiny** as an exemplar of COVID-19 mRNA gene-therapy-vaccination. This was done for two reasons. Firstly, because it was the leading vaccine used by the Ministry of Health and government in New Zealand (i.e., my home country), whose vaccination policies, mandates, and campaigns were hugely controversial, nationally divisive, and caused a national outcry linked to unprecedented community harm essentially explained as unattributable to COVID-19 vaccination (i.e., preexisting medical conditions). Secondly, Comirnaty was associated with the highest number of deaths and hospitalizations in the USA in the first 12 months since its EUA approval by the Food and Drug Administration (FDA). The FDA was a crucial focus because it first approved Comirnaty as the first vaccine for government use and exerted a global influence.

Various FDA (USA), European Medicines Agency (EMA, EU), and Therapeutic Goods Administration (TGA, Australia) regulator-provided (cited in section 1.4) and other specifically cited documents supporting Comirnaty's EUA approval were reviewed for a broader safety understanding independent of New Zealand's Medsafe and Ministry of Health assessments. This was done so New Zealand's MoH leaders, government and politicians, academics, healthcare, and other stakeholders could more broadly understand what has arisen overseas and within New Zealand beyond the invoked narrative.

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You will find a **fundamental difference** in the vaccinated risk-related conclusions of these analytical summaries versus the efficacy and safety narratives provided by these governments (sections 1.1.2-4). These differences are **fully reconcilable** when you comprehend the significant **numerator and denominator biases** evident in these healthcare agencies' calculable unvaccinated COVID-19 case rates (i.e., infections, hospitalizations, and deaths). These evident biases essentially eliminated the negative vaccine effectiveness in the underlying data (section 1.1.5). To remove these biases, I dis-aggregated the 2021-2022 cases from their cumulative totals (2020) using archived web data, used the most recent government population estimates/census data to derive the residual unvaccinated population totals (2021), and then calculated period-specified crude cumulative case rates.

The **purpose** of these analyses was to prove there were important population level safety signals evident in government vaccine surveillance and pharmacovigilance data and share this evidence-based information to **catalyze** (1) a broader awareness of this New Zealand and overseas evidence for negative vaccine effectiveness, vaccine failure, and toxic vaccine lots, (2) scrutiny of the statistical biases evident in the MoH and other healthcare agencies' calculable unvaccinated COVID-19 case rates, which essentially eliminated the negative vaccine effectiveness signal, (3) clinical research into COVID-19 antibody-dependent enhancement of virus infection (ADE), vaccine-associated enhanced disease (VAED), and antigenic imprinting in the New Zealand population, and (4) clinical research into the role of COVID-19 vaccination in *exacerbating* comorbidities most frequently associated with serious-severe COVID-19 outcomes. After all, these safety-harm issues would **predictably manifest** for any coronavirus spike protein-based vaccine targeting critical physiological receptors lining blood vessels and vital organs during pandemic waves associated with **antigenically distinct strains and mass vaccination** (sections 1.1.6-9).

1.1 Analysis of Government COVID-19 Surveillance Data Demonstrates Negative Vaccine Effectiveness (Global and National Data)

1.1.1 High Rates of COVID-19 Vaccination Quadrupled and Tripled Global Rates of COVID-19 Infection and Death Respectively Over Low Vaccination Rates

The bottom line: Nations that achieved high rates of COVID-19 vaccination experienced significantly higher COVID-19 infection and death rates than nations achieving lower vaccination rates. Analysis of Our World in Data (OWID) demonstrated that high rates of COVID-19 vaccination were associated with significantly higher weighted mean infection rates per million (4.0x), death rates per million (3.2x), and vaccination rates per 100 population (4.6x) compared with low vaccination rate nations. The observed proportion of COVID-19 infections and associated deaths was larger in high vaccination rate nations and smaller than expected in low vaccination rate nations. These group differences were highly significant.

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The OWID data (to 31/12/21) comprised 77 nations, 4.5 billion doses, 2.3 billion people vaccinated, 3.9 billion population, 227 million cases diagnosed, and 4.1 million deaths.[3] These 77 nations provided complete datasets for relevant parameters (i.e., total cases and deaths per million- and total people vaccinated per hundred- of the population), which were organized into high and low vaccination rate groups (Group-1: N = 57 countries, ≥50 per 100 population. Group-2: N = 20 countries, <50 per 100 population). Group weighted mean COVID-19 infection and death rates per million and population proportions were compared using Welch's unpaired T-test and Chi-square test of independence, respectively.

There was a weighted mean of 65,202 (SD = 55,318, standard deviation) compared with 16,440 (SD = 29,770) *infections per million* of population, which was associated with a weighted mean of 66.8 (SD = 9.3) and 14.6 (SD = 13.3) people vaccinated per 100 of population, for Group-1 and -2 respectively (*Welch's unpaired T-test, infections per million difference, t (62) = 4.9, 2-tailed p < .00001*). The observed proportion of COVID-19 infections was higher in Group 1 (high vax-rate), and lower in Group 2 than expected, and these group differences were highly significant [*Chi-square test of independence,* X^2 *(df = 1, N = 3,877,605,243) = 19,818,764, p < .00001*]. These results indicate that high vaccination rates were associated with significantly higher COVID-19 infection rates and population proportions than expected compared with low vaccination rate nations.

There was a weighted mean of 1,174 (SD = 1,094) compared with 368 (SD = 703) COVID-19-associated *deaths per million* of the population for Group-1 and -2, respectively (*Welch's unpaired T-test, COVID-19 deaths per million difference,* t (52) = 3.8, 2-tailed p < .0004). The observed proportion of COVID-19-associated deaths was higher in Group 1 (high vax-rate), and lower in Group 2 than expected, and these group differences were highly significant [*Chi-square test of independence, X2 (df = 1, N = 3,877,605,243) = 280,763, p < .00001*]. These results demonstrate that high vaccination rates were associated with significantly higher COVID-19-associated death rates and population proportions than expected compared with low vaccination rate nations.

The results detailed above were corroborated via a published *causal impact analysis*, which compared the before and after vaccination impact on infection and death rates to November 2021 (OWID data).[4] This study showed that COVID-19 vaccination had a statistically significant strong propensity to causally increase deaths per million (y1) and infections per million (y2) over what would have been expected without vaccination. Y1 (deaths) comprised 128 countries, with a country rates increase/decrease ratio of +115/-13 and an average causal impact of +463%. Y2 (infections) included 103 countries and showed a country rates increase/decrease ratio of +105/-16 and an average causal impact of +261%.

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1.1.2 COVID-19 Vaccination Increased COVID-19 Infection Rates Over the Unvaccinated

The bottom line: COVID-19 vaccination did not prevent SARS-CoV-2 infection. On the contrary, in general, the COVID-19 infection rates were significantly higher in the 1-, 2-, and 3-dose COVID-19 vaccinated than in the unvaccinated.

New Zealand: The New Zealand Ministry of Health (MoH, from 22/02/22 to 4/7/22, ≥12yr demographics.[5] Statistics New Zealand.[6]) data shows their COVID-19 vaccination strategy did not protect the population from COVID-19 infection as originally touted but instead significantly increased the risk and rates of COVID-19 infection for all vaccine dose groups compared with the unvaccinated. The New Zealand MoH data shows the COVID-19 vaccinated population (1-3 doses) accounted for 96% of cumulative COVID-19 infections while accounting for 93.4% of the ≥12yr population (NZ Stats: 4,345,230). There were a cumulative 7,311, 16,222, and 8,608 more COVID-19 infections per 100,000 in the 1-, 2-, and 3-dose vaccinated, respectively, than the unvaccinated. This corresponded with higher rates of COVID-19 infections in the 1-dose (1.5x), 2-dose (2.0x), and 3-dose (1.5x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 infections was higher in the 1-, 2-, and 3-dose vaccinated and lower in the unvaccinated than expected. These differences were highly significant for all vaccine dose groups (Chi-square test of independence, all $p < .00001$). This data indicates that the 1-, 2-, and 3-dose vaccinated groups experienced a significantly increased risk of COVID-19 infection compared with the unvaccinated.

England: The UK Health Security Agency (UKHSA) vaccine surveillance data showed its vaccination strategy did not prevent SARS-CoV-2 infection in the England population (i.e., Omicron). Instead, this vaccination strategy (1-, 2-, and 3-doses) significantly increased the rates, proportions, and absolute risk of infection in vaccinated working-age adults (18-59yrs) and the elderly (≥60yrs) over the unvaccinated. The 2022 UKHSA data was analyzed between 08/11/2021 and 31/03/2022 (i.e., report 49 2021 - Report 13 2022).[7,8] This analysis was done using rates calculated from the raw COVID-19 case data and the vaccinated and population totals because the UKHSA's "unadjusted" COVID-19 infection, hospitalization, and death rate data for the vaccinated were significantly and non-uniformly altered over that calculable from the raw data. In contrast, their unvaccinated COVID-19 rates were broadly as calculated.

The vaccinated accounted for most COVID-19 infections (73%), with vaccinated working-age adults accounting for the highest percentage of total infections (57%). There were 4,927, 20,516, and 3,396 more COVID-19 infections per 100,000 in the 1-, 2-, and 3-dose vaccinated working-age adults, respectively than the unvaccinated (18-59yrs) and 2,835, 33,566, and 1,928 more COVID-19 infections per 100,000 in 1-, 2-, and 3-dose vaccinated elderly than the unvaccinated (≥60yrs). This corresponded with a higher rate of COVID-19 infection in working-age vaccinated adults (1-dose 1.6x, 2-dose 3.5x, and 3-dose 1.4x) and in the vaccinated elderly (1-dose 1.8x, 2-dose 10.1x, and 3-dose 1.5x) compared with the unvaccinated.

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There were 4,757 more infections per 100,000 in 1-dose vaccinated kids-youth compared with the unvaccinated (<18yrs), which corresponded with a 1.3x higher rate of infection over the unvaccinated. Vaccinated infection proportions were higher than and unvaccinated proportions lower than expected for working-age adults and the elderly (1-, 2-, and 3-doses) and kids-youth (1-dose), and these differences were highly significant (Chi-square test of independence, all $p < .00001$). In other words, COVID-19 vaccination failed to protect against COVID-19 infection as initially touted by the UK government, but instead, it significantly increased the risk of infection over the unvaccinated.

Scotland: The Public Health Scotland (PHS,[9] Mid-2021 population estimates.[10]) data shows the vaccinated population (1-3 doses) accounted for 80.6% of all COVID-19 infections while accounting for 78.6% of the population. There were 2,780 and 5,599 more COVID-19 infections per 100,000 in the 1- and 2-dose vaccinated, respectively, and 2,063 fewer COVID-19 infections per 100,000 in the 3-dose vaccinated than the unvaccinated. This corresponded with higher rates of COVID-19 infections in the 1-dose (1.3x), and 2-dose (1.7x) vaccinated and a lower rate in the 3-dose vaccinated (0.74x) compared with the unvaccinated. The observed proportion of COVID-19 infections was higher in the 1- and 2-dose vaccinated and lower in the unvaccinated than expected, with this observed-expected proportion difference being reversed (i.e., vaccinated-lower, unvaccinated-higher) with the 3-dose vaccinated (Chi-square test of independence, all $p < .00001$). This data indicates that the 1- and 2-dose vaccinated experienced an increased risk (i.e., cumulative rate and proportion) of COVID-19 infection over the unvaccinated. At the same time, a third dose temporarily ameliorated this enhanced infection risk (i.e., for a duration less than the booster interval).

Canada: The Public Health Agency of Canada data (PHAC)[11] shows the COVID-19 vaccinated population (1-3 doses, ≥5yr demographics) accounted for 84.8% of cumulative COVID-19 infections while accounting for 71.1% of the population (Statistics Canada).[12] There were a cumulative 211, 620, and 756 more COVID-19 infections per 100,000 in the 1-, 2-, and 3-dose vaccinated, respectively, than the unvaccinated. This corresponded with higher rates of COVID-19 infections in the 1-dose (1.4x), 2-dose (2.2x), and 3-dose (2.4x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 infections was higher in the 1-, 2-, and 3-dose vaccinated and lower in the unvaccinated than expected. These differences were highly significant (Chi-square test of independence, all $p < .00001$). This data indicates that the 1-, 2-, and 3-dose vaccinated groups experienced an increased risk (i.e., cumulative rates and proportions) of COVID-19 infection compared with the unvaccinated.

1.1.3 COVID-19 Vaccination Increased the Risk of COVID-19 Death Over the Unvaccinated

The bottom line: At the national level during the Omicron wave, there was a significant COVID-19 death prevention disbenefit or no benefit to COVID-19 vaccination across the various dose and demographic

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categories at the national level. Government claims (in general) that COVID-19 vaccination prevented COVID-19 death despite enhanced infection rates are unsupported by the majority of its data, especially in the elderly, who accounted for most of the COVID-19 death burden (UKHSA, 90%).

England: The UKHSA COVID-19 death data showed there was a zero-to-negligible COVID-19 death prevention benefit to COVID-19 vaccination in kids, youth, and working-age adults over the unvaccinated (1-, 2- and 3-doses), while the elderly vaccinated accounted for most of the COVID-19 deaths within 28 days of a positive COVID-19 test.[13,14] The elderly vaccinated (≥60yrs, 1-3 doses) accounted for 76.5% and the unvaccinated elderly 13.6% of all COVID-19 deaths, while the elderly accounted for 23% of the England population. There were 48 and 451 more COVID-19 deaths in the 1- and 2-dose vaccinated elderly, respectively, and 216 fewer in the 3-dose elderly vaccinated than the unvaccinated elderly. This corresponded with a 1.2x and 2.7x higher rate and 0.2x lower rate of COVID-19 death in the 1-, 2, and 3-dose vaccinated, respectively, compared with the unvaccinated. The unvaccinated COVID-19 death proportions were lower than and vaccinated COVID-19 death proportions higher than expected in the 1- and 2-dose elderly populations, with this observed-expected proportion difference being reversed (i.e., unvaccinated-higher, vaccinated-lower) for the 3-dose vaccinated elderly (Chi-square test of independence, all $p < .002$). This elderly data indicates 1- and 2-dose vaccination increased the risk of COVID-19 death while a third dose temporarily ameliorated this COVID-19 death disbenefit (i.e., until immunity waned).

Vaccinated kids and youth accounted for 0.04% and unvaccinated kids and youth 0.11% of all COVID-19 deaths, respectively, while accounting for one-fifth of England's population. In other words, the risk of COVID-19 death in those <18yrs was comparatively very low. At peak immunity, there was one more death per million in the 1- and 2-dose vaccinated kids-youth demographic and two fewer deaths per million in the 3-dose vaccinated group (<18yrs), which corresponded with a 1.4x and 1.5x higher rate of COVID-19 death in the 1- and 2-dose vaccinated kids-youth. The working-age vaccinated adults (1-3 doses) accounted for 5.7% and the unvaccinated working-age adults 4.1% of all COVID-19 deaths (18-59yrs) while accounting for 57% of the population. There were 1.8, 1.4, and 7.1 fewer deaths per 100,000 working-age adults, respectively, than the unvaccinated, which corresponded with a 1- and 2-dose COVID-19 death rate of 0.8x and a 3-dose COVID-19 death rate of 0.2x that of the unvaccinated. In working-age adults, the unvaccinated death proportions were higher than and vaccinated death proportions lower than expected for 1-3-doses (Chi-square statistic, all p-values $< .02$). In other words, at the same time, vaccination enhanced the rates and risk of COVID-19 infection in working-age adults it reduced the rates of COVID-19 death (for now) relative to the unvaccinated within 28 days of a positive COVID-19 test.

Scotland: The Public Health Scotland (PHS,[15] Mid-2021 population estimates.[16]) data shows the vaccinated population (1-3 doses) accounted for 83.9% of all COVID-19 deaths while accounting for 78.5% of the

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total population. There were 11 and 3 more COVID-19 deaths per 100,000 in the 2- and ≥3-dose vaccinated, respectively, compared with the unvaccinated. This corresponded with higher rates of COVID-19 deaths in the 2-dose (1.9x) and 3-dose (1.2x) vaccinated. The observed proportion of COVID-19 deaths was higher in the 2- and ≥3-dose vaccinated and lower in the unvaccinated than expected, and this difference was significant at the $p < .05$ level for both 2- and ≥3-dose vaccinated groups (Chi-square test of independence, 2-dose $p = < .00001$, ≥3-dose $p = 0.047$). This data indicates a significant disbenefit to vaccination on COVID-19 death rates and proportions for the fully vaccinated and those receiving ≥3-doses compared with the unvaccinated.

Canada: The Public Health Agency of Canada COVID-19 death data (PHAC, see infection data citation, Table 2, and Statistics Canada[17]) shows the COVID-19 vaccinated population (1-3 doses) accounted for 71.5% of cumulative COVID-19 deaths while accounting for 71.1% of the population. Vaccination provided a *marginal* COVID-19 death prevention benefit (1- and 2-doses) and a disbenefit (3-doses). There were 1.2 more COVID-19 deaths per 100,000 with the 3-dose vaccinated than the unvaccinated, and 2.2 and 0.5 fewer COVID-19 deaths per 100,000 with the 1- and 2-dose vaccinated, respectively. This corresponded with a higher rate of COVID-19 deaths in the 3-dose vaccinated group (1.1x) and lower rates of COVID-19 death in the 1-dose (0.81x) and 2-dose vaccinated (0.96x) compared with the unvaccinated. The observed proportion of COVID-19 deaths was higher in the 3-dose vaccinated and lower in the unvaccinated than expected, with this observed-expected proportion difference being reversed (i.e., vaccinated-lower, unvaccinated-higher) with the 1- and 2-dose vaccinated. These differences were significant for the 3- and 1-dose groups (Chi-square test of independence, 1-dose $p = .04$, 2-dose $p = .28$, 3-dose $p = .01$). This data indicates the 3-dose vaccinated experienced a significantly increased risk of COVID-19 death compared with the unvaccinated (i.e., rates and proportions).

1.1.4 COVID-19 Vaccination Increased the Risk of COVID-19 Hospitalization Over the Unvaccinated

The bottom line: At the national level during the Omicron wave, there was a significant COVID-19 hospitalization prevention disbenefit or no benefit to COVID-19 vaccination across the various dose and demographic categories. Government claims (in general) that COVID-19 vaccination prevented COVID-19 hospitalization despite enhanced infection rates are unsupported by the majority of its data, especially in the elderly, who accounted for the majority of the COVID-19 hospitalizations (UKHSA, 54%).

New Zealand: The New Zealand Ministry of Health data (MoH, see COVID-19 infection data citation) shows the COVID-19 vaccinated population (1-3 doses) accounted for 89.4% of cumulative COVID-19 hospitalizations while accounting for 93.4% of the ≥12yr population (NZ Stats: 4,345,230). There were a cumulative 66 more COVID-19 hospitalizations per 100,000 in the 1-dose vaccinated, and 105 and 239

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fewer hospitalizations per 100,000 for the 2- and 3-dose vaccinated, respectively, than the unvaccinated. This corresponded with a higher rate of COVID-19 hospitalization in the 1-dose (1.1x) and a lower rate in the 2-dose (0.8x) and 3-dose (0.5x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 hospitalizations was higher in the 1-dose vaccinated and lower in the unvaccinated than expected, with this proportion difference, reversed (i.e., vaccinated-lower, unvaccinated-higher) for the 2- and 3-dose vaccinated (Chi-square test of independence, 1-dose $p = .047$, 2- and 3-dose $p < .00001$).

However, in the second half of this period (03/05/22 to 04/07/2022), there were a cumulative 27 and 10 more hospitalizations per 100,000 in the 1- and 2-dose vaccinated, which corresponded with a higher rate of COVID-19 hospitalization in the 1-dose (1.2x) and 2-dose (1.1x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 hospitalizations was higher in the 1- and 2-dose dose vaccinated and lower in the unvaccinated than expected, but these differences were not statistically significant (Chi-square test of independence, 1-dose $p = .18$, 2-dose $p = .23$). This data potentially indicates that during the early phase of the Omicron wave, before vaccinee immunity had waned, there was a modest COVID-19 hospitalization prevention benefit for the 2- and 3-dose vaccinated, however, there was an increased risk of hospitalization with the 1-dose vaccinated. However, as the Omicron wave progressed and immunity waned, there was no COVID-19 hospitalization prevention benefit at best, and at worst a disbenefit, for the 1- and 2-dose vaccinated compared with the unvaccinated, while the relative risk increased for the 3-dose vaccinated from 0.5x to 0.8x.

England: The UKHSA COVID-19 data showed a modest-large COVID-19 hospitalization disbenefit in the 1- and 2-dose elderly vaccinated and a negligible-modest COVID-19 hospitalization prevention benefit to COVID-19 vaccination in kids, youth, and working-age adults over the unvaccinated (1-, 2- and 3-doses).[18,19] The elderly vaccinated (≥60yrs, 1-3 doses) accounted for 45.7%, the unvaccinated elderly 8.1% of all COVID-19 hospitalizations, while the elderly accounted for 23% of England's population. There were 28 and 532 more COVID-19 hospitalizations in the 1- and 2-dose vaccinated elderly, respectively, and 360 fewer hospitalizations in the 3-dose elderly vaccinated than the unvaccinated elderly. This corresponded with a 1.1x and 2.1x higher rate and 0.2x lower rate of COVID-19 hospitalization in the 1-, 2, and 3-dose elderly vaccinated, respectively, compared to the elderly unvaccinated. The unvaccinated elderly COVID-19 hospitalization proportions were lower than expected, and the 1- and 2-dose elderly vaccinated COVID-19 hospitalization proportions were higher than expected, with this proportion difference reversed (i.e., unvaccinated-higher, vaccinated-lower) for the 3-dose elderly vaccinated (Chi-square test of independence, 2- and 3-dose $p < .00001$, 1-dose $p = .16$). This elderly vaccinated data indicates 1- and 2-dose vaccination increased the risk of COVID-19 hospitalization. At the same time, a third dose temporarily ameliorated this COVID-19 hospitalization disbenefit (i.e., temporarily).

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Vaccinated kids and youth accounted for 0.8% and unvaccinated kids-youth 9.0% of all COVID-19 hospitalizations while accounting for one-fifth of England's population. At peak immunity, there were 24, 35, and 34 fewer hospitalizations per 100,000 in the 1-dose, 2-dose, and 3-dose vaccinated kids-youth compared with their unvaccinated demographic, which corresponded with a 0.5x, 0.2x, and 0.3x rate of COVID-19 hospitalization compared with the unvaccinated. The working-age vaccinated adults (1-3 doses) accounted for 22.8% and the unvaccinated working-age adults 13.5% of all COVID-19 hospitalizations (18-59yrs) while accounting for 57% of the population. There were 8.8, 8.2, and 61 fewer COVID-19 hospitalizations in working-age adults per 100,000, respectively than the unvaccinated. This corresponded with a 1- and 2-dose COVID-19 hospitalization rate of 0.9x and a 3-dose rate of 0.3x that of the unvaccinated. In working-age adults, the unvaccinated COVID-19 hospitalization proportions were higher than and vaccinated COVID-19 hospitalization proportions lower than expected for 1-3 doses (Chi-square test of independence, all p-values < .0005). In other words, while vaccination enhanced the rates and risk of COVID-19 infection in working-age adults, it reduced the rates of COVID-19 hospitalization (for now) relative to the unvaccinated within 28 days of a positive COVID-19 test.

Scotland: The Public Health Scotland (PHS,[20] Mid-2021 population estimates[21]) data shows the vaccinated population (1-3 doses) accounted for 79.1% of all COVID-19 hospitalizations while accounting for 77.3% of the total population. There were 5, 25, and 6 more COVID-19 hospitalizations per 100,000 in the 1-, 2-, and 3-dose vaccinated, respectively. This corresponded with higher rates of COVID-19 hospitalizations in the 1-dose (1.1x), 2-dose (1.2x), and 3-dose (1.1x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 hospitalizations was higher in the 1-3 dose vaccinated and lower in the unvaccinated than expected, and this difference was significant at the $p < .05$ level for the 2-dose vaccinated (Chi-square test of independence, 1-dose $p = 0.45$, 2-dose $p = < .00001$, 3-dose $p = 0.09$). This data indicates a marginal-modest disbenefit to vaccination on COVID-19 hospitalization rates and proportions for all vaccine dose groups compared with the unvaccinated, which was significant for the 2-dose vaccinated group proportions.

Canada: The Public Health Agency of Canada data (PHAC, see infection data citation, Table 2, and Statistics Canada[22]) shows the COVID-19 vaccinated population (1-3 doses) accounted for 74.2% of cumulative COVID-19 hospitalizations while accounting for 71.1% of the population. There were a cumulative 8.3, 3.6, and 14.9 more COVID-19 hospitalizations per 100,000 in the 1-, 2-, and 3-dose vaccinated, respectively, than the unvaccinated. This corresponded with higher rates of COVID-19 hospitalizations in the 1-dose (1.1x), 2-dose (1.1x), and 3-dose (1.3x) vaccinated compared with the unvaccinated. The observed proportion of COVID-19 hospitalizations was higher in the 1-, 2-, and 3-dose vaccinated and lower in the unvaccinated than expected. These differences were highly significant (Chi-square test of independence, all $p < .0007$). This data indicates the 1-, 2-, and 3-dose vaccinated groups

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experienced a significantly increased risk of COVID-19 hospitalizations compared with the unvaccinated (i.e., cumulative rates and proportions).

1.1.5 Statistical Bias Evident in Healthcare Agencies' Calculable COVID-19 Case Rates Essentially Eliminated the Negative Vaccine Effectiveness Harm Signal (Data)

The bottom line: This section details the significant numerator and denominator biases evident in all of these healthcare agencies' calculable unvaccinated COVID-19 infection, hospitalization, and mortality case rates (i.e., New Zealand, Scotland, Canada), or in the supposedly "unadjusted" rates they provided (England). These evident biases essentially eliminated the underlying negative vaccine effectiveness or vaccine failure and thus obscured the vaccine-induced harm at the national level.

Four main methods were evident by which bias manifest, including the: (1) provision of national healthcare database population totals that underestimated the total population relative to the most recent Government estimates/census, from which a residual underestimated unvaccinated population total was calculable (i.e., New Zealand, Scotland) (**denominator bias**), (2) use of cumulative case totals that bundled 2020-2021 cases arising before high vaccination rates into the 2022 unvaccinated data (i.e., Canada, New Zealand) (**numerator bias**), (3) provision of vaccinated demographic rates of infection and disease as "unadjusted" that had been non-uniformly altered without specifying their reasons and assumptions (i.e., England) (**altered "unadjusted" rates**), (4) use of vaccinated and unvaccinated definitions that failed to reflect ADE biology and its impact on early (i.e., first dose) and late (i.e., waned immunity) infection, hospitalization, and death risk (i.e., all nations) (**definition bias**).

Any discussion on enhanced rates of COVID-19 infection and disease before its dismissal as inherent bias consequent to vaccinated and unvaccinated group differences by healthcare agencies (i.e., *social behavior interactions, testing behaviors, vaccination prioritization, natural immunity, etc.*), in my view, must first and foremost reflect **three more dominating rate-critical issues**. Firstly, the significant numerator and denominator bias in evidence as summarized above and detailed in section 1.1.5.1. Secondly, the three decades of scientific evidence of **antibody-dependent enhancement of virus infection** (ADE) common to **three other coronaviruses** and their spike protein-based vaccine prototypes means ADE should have been at the forefront of explanations (section 1.1.6). In my view, this ADE differential diagnosis should have ensured that this phenomenon was a key healthcare agency **priority** for clinical research and an important issue in **informed consent guidelines**. Thirdly, the damning evidence that certain Governments and their affiliates had sustainedly invested vast resources in **gain-of-function** genetic modification of coronavirus spike proteins to specifically bypass the need for a zoonosis and enhance human infection and disease rates while then working to censor-suppress its role in the origin of the COVID-19 pandemic (Part-2).

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The above overview gives a broader context specifically to the England and Scotland healthcare agencies' argument structuring and highly conspicuous buttressing. In the final reports in which the UKHSA (31/03/2022[23]) and PHS (16/02/2022[24]) provided COVID-19 cases by vaccination status, they both emphatically cautioned the public not to use their data for vaccine effectiveness calculations. Instead, the UKHSA referred people to Table 5, and the PHS referred people to UKHSA reports 4 and 6,[25] among other publications. These reports provided a list of largely non-peer-reviewed vaccine efficacy publications that heavily biased Alpha and Delta strains using relatively small and/or highly selected populations that failed to represent the national-level outcomes (i.e., **confirmation bias**). Their **argument buttressing was conspicuously devoid** of any discussion on the multi-decade foundational biology of coronavirus ADE and more generally, about antigenic imprinting to explain the negative vaccine effectiveness and vaccine failure, respectively. In my view, historical vaccine effectiveness publications are **irrelevant when discussing current data** for coronaviruses in the face of ADE and antigenic imprinting, both manifested by new strains that are **antigenically distinct** from the original vaccine strain (section 1.1.6-8).

1.1.5.1 Significant Numerator and Denominator Bias Evident in Healthcare Agency Calculable Unvaccinated COVID-19 Case Rates

The following details the significant numerator and denominator bias evident in government healthcare agencies' calculable COVID-19 case rates, which essentially eliminated the negative vaccine effectiveness harm signal and vaccine failure in need of urgent investigation (i.e., ADE, VAED, and antigenic imprinting).

New Zealand (Ministry of Health, MoH): The MoH provided its data as cumulative totals since 26th February 2020, necessitating disaggregation of the Omicron wave data using the web archived data to prevent numerator bias in any spot rate calculations. The MoH provided its Health Service User (HSU 2020[26]) population estimates for the ≥12yr population total (i.e., 4,209,057 on 01/07/2020),[27] not the more recent and larger Statistics New Zealand (NZ-Stats) ≥12yr population estimates (4,345,230 on 31/12/21),[28] from which the residual unvaccinated population total was calculable. Which population total one uses for calculating the residual unvaccinated population is critical given the extremely high vaccination rates in New Zealand. This issue dominates any discussion on statistical bias. The MoH's provision of the HSU 2020 ≥12yr population total in its tables ("Vaccination uptake by ethnicity") effectively halved the residual unvaccinated population compared with NZ Stats. This would double the calculable crude unvaccinated rates and thus **eliminate negative vaccine effectiveness** in all but the 2-dose vaccinated.

The average weekly residual unvaccinated population between March 01 and July 04, 2022, was 288,322 derived using the NZ-Stats ≥12yr population total minus the COVID-19 Immunization Register (CIR) vaccinated total (i.e., NZ-Stats-minus-CIR-all-doses) and 152,149 using the HSU ≥12yr population total minus the CIR vaccinated total (i.e., HSU-minus-CIR-all-doses). During the brunt of the Omicron wave

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and study period, there were 45,309 cumulative new COVID-19 infections in the ≥12yr unvaccinated population yielding an unvaccinated cumulative rate per 100,000 of 15,715 (NZ-Stats-minus-CIR-all-doses) and 29,779 (HSU-minus-CIR-all-doses). As such, the crude unvaccinated cumulative rate was increased by a **factor of 1.9** over the rates derived using the NZ-Stats population. The 1-, 2-, and 3-dose cumulative infection rates were 23,026, 31,937, and 24,323 per 100,000, respectively. The cumulative rate ratios for the 1-dose were 0.8x (NZ-Stats 1.5x), 2-doses 1.1x (NZ-Stats 2.0x), and 3-doses 0.8x (NZ-Stats 1.5x). I concluded that the provision of HSU2020 eliminated the negative vaccine effectiveness in need of **urgent MoH investigation**. This helps explain why the National Immunization Programme website shows **no funding has been awarded** to investigate the predictable antibody-dependent enhancement of virus infection, vaccine-associated enhanced disease, or antigenic imprinting in New Zealand thus far.[29]

Of **great concern** with regards to denominator bias in calculable COVID-19 rates was that between August 04[30] and 11.59 pm August 08, 2022, the MoH switched from HSU2020 to HSU2021,[31] resulting in its 12+ population total increasing from 4,209,057 to 4,452,797 **(+243,740 people)**, which then exceeded the NZ-Stats 2021 12+ population by 107,567 people. By recalculating COVID-19 case rates using both HSU2020 and 2021 populations between 01/03/2022 and 04/07/2022 (i.e., my main study period), the crude unvaccinated cumulative infection and hospitalization rates were **2.6 times greater** using HSU2020 than HSU2021. By using HSU2020, the negative vaccine effectiveness for COVID-19 infection and hospitalizations were **essentially eliminated** in all but the 2-dose infection group. Whereas **pronounced negative vaccine effectiveness** was evident for COVID-19 infections and hospitalizations in **all doses** except the 3-dose hospitalization group using HSU2021. What a difference a few months makes.

The COVID-19 infection rate ratios for the 1-, 2-, and 3-dose vaccinated were 0.8x (2.0x), 1.1x (2.8x), and 0.8x (2.1x) respectively using HSU2020 (no brackets) versus HSU2021 (in brackets). The COVID-19 hospitalization rate ratios (RR) for the 1-, 2-, and 3-dose vaccinated were 0.6x (1.6x), 0.4x (1.1x), and 0.3x (0.7x) respectively using HSU2020 (no brackets) versus HSU2021 (in brackets). An RR > 1.0 indicates negative vaccine effectiveness, along with other measures (i.e., a -ARR and Chi-Square observed-v-expected proportion differences). These rate ratios corresponded with 11,581, 20,492, and 12,878 **more** COVID-19 infections in the 1-, 2-, and 3-dose vaccinated, respectively, and 195 and 23 more hospitalizations in the 1- and 2-dose vaccinated, and 110 fewer COVID-19 hospitalizations in the 3-dose vaccinated, per 100,000, over the unvaccinated by using HSU2021. Had the MoH provided the HSU2021 population total during the brunt of the Omicron wave it would have been highly evident (i.e., more than with NZ Stats) there was a problem with negative vaccine efficacy in preventing COVID-19 infections and hospitalizations. In consequence, New Zealanders were **not emphatically warned of the life-long health risks** of antibody-dependent enhancement of virus infection during their vaccination informed consent. At the same time, doctors who knew about these issues were threatened with medical deregistration for not

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following government guidelines (i.e., NZDSOS).

Of **serious concern** is that the MoH provided the HSU2020 population total knowing its shortcomings (see Excel page "HSU Population" summary table).[32] The MoH confirmed the HSU total was not a total population estimate because it included only people who received health services or were PHO enrolled in a given year only. The HSU was known to miss highly marginalized groups and young people aged 15-45 years, especially males and people of Asian and MELAA ethnicity, whereas COVID-19 does not miss anyone. This would make any residual unvaccinated population calculations using HSU totals extremely sensitive to these deficiencies. In my view, it should have been obvious what the impact would be of using the HSU2020 versus NZ Stats populations on increasing the calculable unvaccinated case rates.

The MoH claimed without providing evidence that the use of the HSU database prevented numerator denominator bias by ensuring the same source of demographic information is used in the numerator and the denominator. As demonstrated above, the provision of the smaller HSU population total created the **significant denominator bias (1.9x or 2.6x)** evident in the calculable unvaccinated rates versus the NZ Stats population total or HSU2021. Given these well-known HSU population shortcomings and their obvious impact on residual unvaccinated COVID-19 rate denominator bias, the **MoH still requested Stats NZ to peer review** the methods used to create the HSU population and its suitability as a denominator for measuring COVID-19 vaccine coverage, and wider use (i.e., **rate calculations**).[33] In my view, that latter act should be a key point for **investigation**.

England (UKHSA): The UKHSA provided vaccinated demographic rates of infection, hospitalization, and death as "unadjusted" that had been **non-uniformly adjusted** without explanation. From reports 49 (2021) to 2 (2022), there was a **large-to-massive disparity** between the provided "unadjusted" case rates and my calculated 2-dose vaccinated COVID-19 infection (>18yr demographics), hospitalization (>30 or 40yr demographics), and death rates (in the >40 or 50yr demographics). The UKHSA significantly reduced their provided vaccinated case rates while leaving the unvaccinated case rates largely unchanged, but even with this act, it was insufficient to hide the rapidly deteriorating 2-dose negative vaccine effectiveness. From week 3 in 2022 the UKHSA then switched from providing 2-dose to ≥3-dose case rates, which removed the major deterioration in 2-dose Omicron infection, hospitalization, and death rates from ready public view.

From week 3 to 13 2022, the ≥3-dose vaccinated COVID-19 infection rates (>18yr demographics) were still significantly higher than the unvaccinated rates, highlighting the negative vaccine effectiveness of ≥3-doses. Reports 3-13, 2022, highlight COVID-19 infection rates in the younger demographics were modified while making no-negligible alterations to the unvaccinated COVID-19 infection rates or the vaccinated and unvaccinated COVID-19 hospitalization and death rates (all demographics). In my view, this lack of alterations in most of the unadjusted rate data validated my crude rate calculation methodology while

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exposing biased-unexplained altered UKHSA rate data. My methodology used the UKHSA's raw case data for COVID-19 infections, hospitalizations, and deaths ("Reports 49-13 Table: *Unadjusted rates of COVID-19 infection, hospitalization, and death in vaccinated and unvaccinated populations*"[34]) and the National Immunization Management Service COVID-19 vaccinated population data as used by the UKHSA (NIMS, "Report Table 49-13: *Provisional cumulative COVID-19 vaccine uptake by age in England*").[35] As of 01/04/22 UKHSA no longer provided case data by vaccination status, making it **impossible** to monitor for evidence of negative vaccine effectiveness and thus *antibody-dependent enhancement of infection*.

Scotland (Public Health Scotland, PHS): The PHS used its Community Health Index dataset, representing those currently registered with a GP practice in Scotland. The PHS declared the limitations of this database for deriving the residual unvaccinated population total but did not alter its rate calculation methodology to mitigate this shortcoming. The PHS data (weekly reports ending 05/11/21 to 11/02/22) displayed a highly variable population total every week and between each of its three data tables within a week (i.e., COVID-19 infections, acute hospitalizations, and deaths). There was also a major unexplained decrease in the unvaccinated population between the report ends 17/12/21 and 31/12/21 without a corresponding increase in the vaccinated population, which had the effect of reducing the total population by circa ten percent in one week. This unjustified act essentially diminished the calculable 2-dose negative vaccine effectiveness.

During my period of assessment (reports ending 05/11/21-11/02/22) there was a mean population total of 5,557,878 (COVID-19 infection tables), 5,442,343 (COVID-19 acute hospitalization tables), and 5,857,333 (COVID-19 death tables), with a minimum-maximum total population difference of 558,948 (infection tables), 848,320 (hospitalization tables), and 20,292 (death tables) within each category, and a minimum-maximum difference of mean population totals between the COVID-19 infection, hospitalization, and death tables of 414,991 – where there should be no difference. Furthermore, there was a **precipitous decrease** in the mean unvaccinated and population totals between the two sub-periods 05/11/21-17/12/21 and 24/12/21-11/02/22, **devoid of explanation**. The mean unvaccinated population declined by 607,949 while the mean vaccinated population correspondingly increased by only 58,125, resulting in a mean population decline of 549,824 (COVID-19 infection tables). Similarly, there was a mean decrease in the unvaccinated, vaccinated, and total populations of 717,072, 49,381, and 766,452, respectively, between these two sub-periods for the COVID-19 acute hospitalization tables. While the mean total population derived from the COVID-19 death tables was 5,857,333 versus the Scotland mid-2021 census population estimate of 5,479,900, the difference between the two sub-periods was only 11,157. In other words, the PHS unvaccinated totals, all PHS-provided age-adjusted rates, and COVID-19 infection, hospitalization, and death rate narratives should be treated with **extreme caution**, in my opinion.

Further compounding this extreme denominator bias, the PHS age-standardized its COVID-19 acute

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hospitalization and death rate data using the aged 2013 European Standard Population (ESP) data. Age standardization is typically used to weight incidence and mortality data to ensure comparability between countries and over time to reflect different population age structures.[36] The PHS justified its use of age standardization for its weekly data by claiming that the unvaccinated were younger than those receiving two or more COVID-19 vaccine doses and that older individuals were more likely to be hospitalized than younger individuals. While vaccination rates were moderately lower in those aged <50yrs by this stage of the pandemic (pg.35),[37] as the UKHSA data demonstrated it was the ≥50-year demographics who dominated COVID-19 deaths (i.e., vaccinated 79%, vaccinated/unvaccinated 96%) and hospitalizations (i.e., vaccinated 53%, vaccinated/unvaccinated 65%), arguably making the need for age standardization a moot point. Scotland could have provided us demographic-specific data like the UKHSA did, which would have provided greater transparency on its data and conclusions. In my view, age standardization was another means for introducing unspecified numerator and denominator bias into rate calculations. The PHS stopped providing case data by vaccination status as of 16/02/22, making it **impossible** to monitor for evidence of negative vaccine effectiveness and thus *antibody-dependent enhancement of infection*.

Canada: The Public Health Agency of Canada (PHAC) provided cumulative case data since 14 December 2020 (i.e., the start of their vaccination campaign) rather than weekly or monthly new case data. Figure 5 in each report ("*Distribution of confirmed COVID-19 cases reported to PHAC by vaccination status as of*," i.e., May 08, 2022[38]) shows the cumulative unvaccinated percentage of COVID-19 cases, hospitalizations, and deaths as 45.0%, 55.9%, and 56.7% respectively, along with the vaccinated percentages. However, when unvaccinated percentages were calculated using the difference between May 08 and April 11 (i.e., new cases in one month), 2022, these percentages become 19.3% (2.3x less), 22.4% (2.5x less), and 30.5% (1.9x less) respectively. I concluded the use of cumulative data since 14/12/20 **biased** higher unvaccinated percentages and rates, which essentially eliminated the negative vaccine effectiveness harm signal.

In Table 3 ("*Risk of severe outcomes among unvaccinated cases, compared to fully vaccinated cases and cases fully vaccinated with an additional dose, April 11, 2022, to May 08, 2022,*" ≥5yr of age) PHAC provided 4-week age-standardized rate ratios for COVID-19 hospitalizations for the 2-dose (3x) and 3-dose (5x), and COVID-19 deaths for the 2-dose (5x) and 3-dose (7x) (i.e., unvaccinated compared to vaccinated). PHAC provided an **associated narrative** stating, "*From April 11, 2022, to May 08, 2022, compared to fully vaccinated cases, unvaccinated cases were 3 times more likely to be hospitalized and 5 times more likely to die as a result of their illness. Compared to cases fully vaccinated with an additional dose, unvaccinated cases were 5 times more likely to be hospitalized and 7 times more likely to die due to their illness, during this same 4-week period (Table 3).*" **However**, according to my analysis, the only way one can approximate the PHAC narrative associated with Table 3 is to calculate rate ratios using the **cumulative data since 14/12/2020** and not new cases between April 11 and May 08, 2022, **as stated in Table 3s' legends**.

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By using the cumulative raw data since 14/12/2020 for rate analysis as of May 08, 2022, then the unvaccinated had a 2.7x and 5.2x higher rate of COVID-19 hospitalization, and a 3.1x and 5.2x higher rate of COVID-19 death than the 2-dose and 3-dose vaccinated respectively. My calculated cumulative rate ratios were similar in outcome to PHAC's age-standardized COVID-19 hospitalization rate ratios, while their age-adjusted COVID-19 death rate ratios were moderately higher (see above). **However**, when the **new cases** between April 11 and May 08, 2022 (i.e., as stated in the Table 3 legend) were used to calculate rate ratios, then the conclusion was **fundamentally different** from that provided by PHAC. That is, the 2-dose and 3-dose vaccinated experienced a 1.1x and 1.7x higher rate of COVID-19 hospitalization and a 0.8x and 1.0x rate of COVID-19 death than the unvaccinated. In other words, PHAC's age-adjusted rates and associated narrative, presumably **derived using the cumulative data** since 14/12/2020, **obscured the higher rates** of COVID-19 hospitalization in the 2- and 3-dose vaccinated and the 3-dose vaccine failure in COVID-19 death prevention (i.e., the COVID-19 death rate ratio was 1.0x the unvaccinated) between April 11 and May 08, 2022. PHAC also failed to communicate the higher rates of COVID-19 infection in the 2- and 3-dose vaccinated (i.e., 1.2x and 2.1x the unvaccinated, respectively). This issue was the same for all Table 3s in the PHAC reports used for rate analysis in sections 1.1.2-4 (March 24,[39] April 29, [40] May 27,[41] 2022).

Case definition bias: a crucially important form of COVID-19 infection rate bias relates to the definition of the unvaccinated and vaccinated, which failed to reflect the biology of ADE and the infection risk impact of low-rising and low-waning levels of antibody immunity (sections 1.1.6.2 and 1.1.7). The UKHSA, PHS, and PHAC defined the vaccinated (2-doses) and boosted (≥3-doses) as those ≥14 days after their second or third/fourth vaccinations, respectively, while **transferring** the <14-day case risk to the previous vaccinated or unvaccinated group. The UKHSA and PHS defined the first dose as those who received one dose ≥21 days before the specimen date (PHAC ≥14 days). The partially vaccinated were those who received one dose before the specimen date (UKHSA <20 days, PHAC <14 days), while the PHS called these **unvaccinated**. The MoH definitions were less clear. In general, these definitions ignore the biology of antibody-dependent enhancement (ADE) of virus infection in which ADE is observed in the presence of low concentrations of non-neutralizing and/or infectivity-enhancing antibodies that one would putatively observe with rising immunity shortly after the first vaccine dose. All countries assessed showed evidence of higher crude rates of COVID-19 infection in the 1-dose vaccinated than the unvaccinated (i.e., England 1.4x, Scotland 1.3x, Canada 1.4x, and New Zealand 1.5x). This suggests these governments' definition of the vaccinated was inappropriate for capturing the gamut of risks against COVID-19 infection in the face of **predictable ADE**.

Furthermore, as a general comment in all nations assessed, the case definitions for COVID-19 death and acute hospitalization fail to reflect an all-cause morbidity and mortality definition. Instead, healthcare

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agencies have isolated a very narrow 28-day window, which is inconsistent relative to their booster date, to assess serious disease outcomes. This assessment window avoided the majority of vaccine-induced toxicity and harm that had already occurred (i.e., *c.50% within two weeks of vaccination, via my VAERS reconnaissance analysis to November 2021*). In my view, any government narrative based on this narrow window is a **best-case contrivance**, which excludes the serious-severe vaccine adverse events and the 21-14-day periods after primary and booster immunizations, respectively, when ADE could arise, and the longer inter-booster period when protective immunity has waned.

1.1.5.2 Healthcare Agencies' Argument Buttressing to Invalidate Negative Vaccine Efficacy

The UKHSA and PHS inform us the vaccination status of cases, hospital inpatients and deaths should not be used to assess vaccine effectiveness because of inherent biases consequent to vaccinated and unvaccinated population differences (i.e., *social behavioral interactions, testing behaviors, vaccination prioritization, and natural immunity*).[42] How this innate bias compares with the denominator and numerator bias evident in government surveillance data or the use of supposedly "unadjusted" rates can't be assessed from their quantitatively unsubstantiated statements of opinion. This section **teases inherent bias apart** focused on the formulas: absolute risk reduction (ARR) = unvaccinated rate – vaccinated rate, and rate ratio (RR) = vaccinated rate / unvaccinated rate. A negative vaccine effectiveness would be indicated by a negative ARR or a RR>1.0.

Social behavior bias: Any negative vaccine efficacy artifact would suggest the unvaccinated engaged in behaviors that lowered their case rates to less than the vaccinated, and/or the vaccinated engaged in behaviors that increased their case rates. This would imply the unvaccinated maintained social distancing and wore masks more frequently, and stayed away from people, public transportation, public events, dense populations, and work. This would also imply that the vaccinated may have believed or trusted their government's narrative that they were protected and thus engaged in risky behaviors that increased their infection rates above the unvaccinated. This would imply they were less stringent in maintaining their social distancing and wearing masks, increased their socialization rates, increased their use rates of public transport, and more frequently visited public superspreader events. **Does this sound right?**

Natural infection bias: The UKHSA and PHS suggested prior infection could have increased background rates of naturally acquired immunity in the unvaccinated, thus lowering unvaccinated case rates to create negative vaccine efficacy. This argument **topples in New Zealand** because our population was still experiencing its first true pandemic wave of community transmission (i.e., not previously infected). Yet, according to my calculations, negative vaccine efficacy was already evident during the initial Omicron wave. In the Northern Hemisphere, nucleoprotein antibody seroprevalence indicative of natural infection confirmed rates increased from 18.1% (UKHSA Report 36, August 2021) to 36% (UKHSA, Report 12,

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February 2022). Yet, the statistically significant negative vaccine efficacy was already evident in August 2021 (UKHSA report 36) and all subsequent reports that disclosed case rates by vaccination status. It **should be noted** these reports were available to the Ministry of Health just after Auckland's August 2021 lockdown and the ensuing mandated and induced national vaccination campaign.

Testing bias: This would imply the unvaccinated were less likely, and/or the vaccinated more likely to be tested (i.e., *even though they were vaccinated and supposedly protected*) while potentially being impacted by their government's use of high false-positive PCR diagnostic methods using cycle thresholds >35 (see section 1.7.2, i.e., bogus case generator). To convert a 2-dose negative vaccine efficacy (-ARR%, RR >1.0) to a vaccine failure (ARR = 0, RR = 1.0), one would need to increase the unvaccinated COVID-19 case rates by 2.4x (England), 1.7x (Scotland), 2.2x (Canada), and 2.0x (New Zealand), meaning testing rates would need to increase significantly more than these case rate multiples. In this scenario, there would have been no benefit to vaccination, **only harm**. Testing bias would also assume the unvaccinated were able to avoid COVID-19 testing (i.e., for work, school, public gatherings, crossing county, and country borders, etc.). While I have no verifiable evidence, claims arose on social media from May 2021 that at least one government healthcare agency not detailed in this specific analysis was using different PCR cycle thresholds between the vaccinated and unvaccinated with vaccinated reinfections. In the fullness of time, it will be important to understand if using different PCR cycle thresholds impacted case rates more widely.

1.1.6 A Biological Explanation for Negative Vaccine Effectiveness and Vaccine Failure Rooted in a Multi-Decade Base of Coronavirus and Vaccine Science

Definitions: Antibody-dependent enhancement of virus infection is a well-described phenomenon associated with coronavirus vaccines targeting the spike protein. In this situation, viral infection is enhanced after vaccination with one strain and upon (re)infection with a different strain. Thus, ADE represents an alternative antibody-specific mechanism of virus infection of cells. A highly focused definition for vaccine-associated enhanced disease (VAED) would involve the modified clinical presentation of infections affecting people vaccinated with one strain (i.e., Wuhan) and exposed to a different strain (i.e., Delta, Omicron, etc.),[43] which can enhance pathogenicity by intensifying the immuno-inflammatory response. However, a broader VAED definition, and one I subscribe to, is that implied by Pfizer's listed array of diseases, organs and tissues, and symptoms under VAED as detailed in Table 5 **footnote a**) (pg.11),[44] which can be summarized as **vaccine-associated pathologies and symptoms** linked to specific organs and tissues including vascular endothelium-related, blood clotting-related, and heart, respiratory, brain, kidney, and gastrointestinal organs, for reasons that will become clear in section 1.3.

Differential diagnoses: It is my view that genuine **negative vaccine efficacy** or vaccine-induced enhanced rates of COVID-19 infection would imply the population's immune response facilitated viral infection (i.e.,

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antibody-dependent enhancement of virus infection or ADE) or the COVID-19 vaccine damaged-corrupted the population's immune system or response thus making people more susceptible to infection (i.e., a type of vaccine-induced AIDS). With immunization for a mutation-prone RNA virus using surface glycoprotein antigens, **vaccine failure** would be expected to result from the **combination and concurrency** of antigenic imprinting (section 1.4.2) and immune escape by an antigenically distinct strain (i.e., Omicron). With **vaccine failure, efficacy would trend to zero** but would not be less than zero.

Importantly, the significant bias evident in calculable COVID-19 case rates resulted in negative vaccine effectiveness being **converted** into vaccine failure or positive effectiveness. All this rate hiding effort, and yet how many clinical **research projects** were funded in New Zealand, England, Scotland, and Canada to study ADE, VAED, and antigenic imprinting during the Omicron wave? A note of caution is also merited: when reading vaccine efficacy and antigenic imprinting publications that use the term breakthrough infections or vaccine failure, it is essential to understand if this was **assumed**,[45] or that ADE and antigenic imprinting had been assessed and eliminated from involvement in those infections.

1.1.6.1 The Majority of Pre-COVID-19 Coronavirus Spike Protein Vaccine Prototype Publications Over 3-Decades Warned About the Vaccine-Induced ADE Risk

There is a **three-decade vaccine industry legacy** of antibody-dependent enhancement (herein "ADE") of virus infection and its related vaccine-associated enhanced disease (VAED), in the human and veterinary vaccine fields associated with **coronaviruses and their spike protein-based** vaccine prototypes in animal studies. A significant body of scientific publications describing coronavirus vaccine-induced mechanisms of ADE and the results of numerous animal challenge and human ex-vivo/in-vitro studies demonstrate the adverse biological effects of ADE/VAED. This coronavirus spike protein vaccine-induced ADE legacy includes studies for SARS-CoV-1 after its emergence in 2002,[46,47,48,49,50,51,52,53,54,55,56,57] Middle East Respiratory Syndrome (MERS),[58,59] and Feline Infectious Peritonitis (FIP).[60,61,62,63,64,65]

There was **ample scientific warning** before the SARS-CoV-2 pandemic written in black and white journal text about the risk of ADE associated with coronavirus spike protein-based vaccines (i.e., *most of the cited SARS and MERS publications*) to have known "**harm was highly probable**" for SARS-CoV-2 spike protein-based vaccines. Eighteen years ago, I used this ADE insight (and highly probable spike protein-ACE2-induced pathologies) to deselect SARS-CoV-1 as a potential vaccine candidate for development and funding acquisition. This sentiment was further reflected in my blog on 13/12/2020,[66] which will become obvious upon reading section 2. Thus, in my long-standing view, the enhanced rates of COVID-19 infection and vaccine-induced disease were **fully predictable**.

In my experience, during the vaccine R&D process, particularly during lead-optimization and before

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entering clinical studies, it is **incumbent on the innovator** to identify known and theoretical risks and propose plans to monitor and mitigate those risks or terminate the program. Before COVID-19 this was an obligatory part of the R&D process required before testing any vaccine in humans. All of those above-cited ADE publications were **readily available** to COVID-19 vaccine company R&D scientists, the National Institutes of Health (NIH) scientific leadership via its extensively funded gain-of-function research (Part-2), the FDA/other drug regulators, and WHO COVID-19 vaccine advisory board experts before the regulatory approval and/or their promotion of genetically modified prefusion-stabilized (i.e., **NIH Technology Transfer**[67]) SARS-CoV-2 spike protein encoded gene-therapy-vaccines.[68,69] Thus, when you search Comirnaty's FDA, EMA, and TGA regulatory review documents for mention of ADE **you will find none**. Given the historical spike protein vaccine ADE legacy, and in consideration of the informed consent process, this was both **conspicuous and ominous by its absence** in my opinion.

1.1.6.2 Post-COVID-19 Discoveries Confirm Biological Factors Associated with SARS-CoV-2 ADE and a Conceptual ADE-Neutralization Threshold

Recent studies utilizing anti-spike monoclonal antibodies and plasma samples obtained from COVID-19 patients highlight numerous mechanisms involved in SARS-CoV-2-associated ADE. These mechanisms involve immune cells like monocytes, macrophages, and B-lymphocytes expressing specific antibody receptors (i.e., *Fc gamma or fragment crystallizable, FcγR, namely FcγRIA, FcγRIIA, and FcγRIIIA*) and complement component receptors (i.e., *C1q-, ubiquitously expressed on cell surfaces, including respiratory epithelial cells*). These ADE mechanisms can be FcγR-dependent but ACE2-independent, FcγR-independent but ACE2-dependent and S-protein conformational change-dependent (i.e., *N-terminal domain infectivity enhancing antibodies*), or both FcR- and ACE2-dependent ADE.[70,71,72,73,74,75,76,77] Increased viral gene or dysregulated host immune gene expression was evident under ADE conditions, signifying ADE was not biologically benign.[78]

In general, the experimental conditions for evaluating ADE in-vitro varied, and typical of biological research, what happens ex-vivo/in-vitro may not always be replicated in-vivo in humans. Nevertheless, a consistent theme emerged showing that ADE appears to operate in a **time-dependent and antibody-concentration-dependent** manner but not in a viral-dose-dependent manner.

SARS-CoV-2 infection induced ADE antibodies, which elicited an ADE profile for at least 6 months post-infection. This ADE was observed only in highly diluted plasma while strong viral neutralization occurred at lower dilutions, indicating ADE-inducing antibodies may function at **lower concentrations** than neutralizing antibodies.[79] SARS-CoV-2 neutralizing activity was detected in most of the IgG-positive sera (i.e., 63% of COVID-19 patient samples), while ADE antibodies were found in more than 40% of acute COVID-19 patients. Neutralizing activity was detected in most IgG-positive sera, but ADE counteracted

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this in sub-neutralizing conditions in the presence of FcγR or complement receptors.[80] Infectivity-enhancing N-terminal domain (NTD) antibodies were also shown to operate in a concentration-dependent manner, inducing an **open conformation** of the receptor binding domain to augment ACE2 binding, but this did not work when neutralizing antibodies were at high levels.[81] Certain monoclonal anti-spike protein antibodies derived from COVID-19-infected subjects and approved for human use also potentially cause ADE in a narrow range of antibody concentrations.[82,83,84,85]

Similarly, sera collected after SARS-CoV-2 spike protein **mRNA vaccination** (i.e., Spikevax, Moderna) had the potential to cause ADE from an early stage and up to at least six months after vaccination. Both neutralizing and ADE of infection were detected, with neutralization demonstrated at high serum concentrations and **ADE at low concentrations**. The ADE was observed within a relatively narrow window of antibody and serum concentrations, with the amount of virus added to the culture unrelated to the development of ADE in the assay.[86]

Severe COVID-19 infections were typically associated with high titers of SARS-CoV-2 spike protein-specific antibodies. The antibody titer was **positively correlated with the severity** of the disease while demonstrating less neutralization potency.[87] A preprint study highlighted that enhancement of SARS-CoV-2 cell entry was more commonly detected in plasma from severely-affected elderly patients with high titers of SARS-CoV-2 spike protein-specific antibodies, which was mediated via the FcγRII receptor.[88] Levels of NTD infectivity-enhancing antibodies were also detectable at high levels in severe COVID-19 patients.[89]

Significance: Collectively, this **ADE time-dependency** or **antibody-concentration-dependency** phenomenon indicates that during the early stages of COVID-19 infection or not until several months post-infection or in the early stages (i.e., low-rising immunity) and months post-vaccination (i.e., low-waning immunity), when neutralizing antibodies are at sub-neutralizing levels or below a putative **ADE-neutralization threshold**, then ADE may facilitate SARS-CoV-2 viral infection and the subsequent course of disease progression via multiple mechanisms.[90,91] The severity of COVID-19 disease may also be linked to ADE **infectivity-enhancing antibodies** and is positively correlated with anti-spike protein antibody titer.

1.1.7 The Biological Features Associated with Antibody-Dependent Enhancement (ADE) of Virus Infection Mirror Vaccine Surveillance Data Outcomes (Results Discussion)

The ADE time-dependency or antibody-concentration-dependency evident in the biological science (in-vitro/ex-vivo data) has putatively **manifested itself** in the New Zealand, England, Scotland, and Canada healthcare agency COVID-19 infection and +/-death data (in-vivo data) once rate biases are removed from the calculable rates. This negative vaccine effectiveness was evidenced by negative absolute risk reduction (-ve ARR), rate ratios >1.0x (RR), and statistically significant observed-v-expected proportion differences.

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692 The negative vaccine effectiveness evident in the first-dose-vaccinated COVID-19 infection rate ratios (i.e.,
693 New Zealand 1.5x, England 1.4x, Scotland 1.3x, and Canada 1.4x) aligns with the biological finding that
694 ADE of infection displays an antibody-concentration-dependency. In other words, as vaccine-induced
695 immunity rises but is still sub-neutralizing or below the ADE-neutralization threshold, the ADE putatively
696 manifests. The UKHSA data best shows how the second dose vaccine effectiveness (-ARR%, RR>1.0)
697 steadily deteriorated over time across all demographics between report week 39 (26/09/2021)[92] and report
698 week 2 (09/01/2022),[93] which was putatively associated with rapidly waning vaccine-induced immunity
699 and the emergence of the antigenically distinct Omicron strain. These waning two-dose results putatively
700 evidence the time- and antibody-concentration-dependency of ADE of infection. All COVID-19 infection
701 rate ratios improved between the second and third doses (i.e., New Zealand 2.0x→1.5x, England 18-59
702 years 3.5x→1.4x and ≥60 years 10.1x→1.5x, and Scotland 1.7x→0.74x) indicating the negative vaccine
703 effectiveness was **ameliorated** with the third-dose. This amelioration supports the antibody-concentration-
704 dependency phenomenon of ADE. In other words, the third dose boosted neutralizing antibody levels from
705 below their sub-neutralizing levels to back above the ADE-neutralization threshold.

706 The England elderly vaccinated (1-3 doses), who accounted for the largest majority of all COVID-19 deaths
707 (77%) and hospitalizations (46%), highlight several interesting ADE-like phenomena. Firstly, the first dose
708 demonstrated negative vaccine effectiveness (Death: RR 1.2x, ARR -0.048%, Hospitalization: RR 1.1x,
709 ARR% -0.028%), which deteriorated approximately 10-fold-plus in the 2-dose elderly vaccinated (Death:
710 RR 2.7x, ARR -0.45%, Hospitalization: RR 2.1x, ARR% -0.53%). Given the analysis period (i.e., Omicron
711 wave, 1-3-doses), this 2-dose data putatively proxied waning immunity. Secondly, a third dose ameliorated
712 this two-dose negative vaccine effectiveness by putatively bringing antibody levels above the ADE-
713 neutralization threshold (Death: RR 0.2x, ARR +0.22%, Hospitalization: RR 0.2x, ARR% +0.36%). This
714 third dose amelioration phenomenon was also apparent in the Scotland data, in which the two-dose rate
715 ratio (1.9x) improved with a third dose (1.2x). However, this was still insufficient to convert a negative into
716 positive vaccine effectiveness at the whole population level.

717 This UKHSA COVID-19 death and hospitalization data also feature a time-dependency or concentration-
718 dependency phenomenon associated with the short first-dose period (i.e., concentration-dependent) or
719 associated with waned immunity in those who failed to complete their primary vaccination and with the
720 two-dose vaccinated (i.e., time-/concentration-dependent). The UKHSA COVID-19 death and
721 hospitalization data, and PHS COVID-19 death data, also highlight a third-dose amelioration (i.e.,
722 concentration-dependent) effect in COVID-19 death rates compared with the two-dose rates. Both time-
723 dependency and amelioration phenomena could indicate the impact of antibody concentrations relative to
724 a putative ADE-neutralization threshold, as discussed in the COVID-19 ADE biology.

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Scotland (i.e., 1.1x, 1.2x*, 1.1x, for 1-, 2-, and 3-doses, respectively) and Canada data (i.e., 1.1x*, 1.1x*, 1.3x*, for 1-, 2-, and 3-doses respectively) also demonstrated higher rates of COVID-19 hospitalizations than the unvaccinated, and thus negative vaccine effectiveness at the population level. The asterisks* indicate that the observed proportions of COVID-19 hospitalizations were higher in the vaccinated and lower in the unvaccinated than expected, and these differences were significant.

This ADE phenomenon **may partially explain** these enhanced rates of COVID-19 hospitalization and death based on an implied time-/concentration-dependency in the 1-, 2-, or 3-dose data. However, any potential ADE effect in the COVID-19 hospital and death rates is **confounded** by other contemporaneous vaccine-associated enhanced disease (VAED) phenomena. These confounding phenomena are putatively linked to an array of virus-free spike protein-related pathologies in furin/ACE2-rich tissues and organs, which overlaps with the most prevalent comorbidities associated with severe COVID-19 outcomes in at-risk populations (i.e., the elderly) (section 1.3.3). Lipid nanoparticle chemical-induced pro-inflammatory responses (section 1.3.2) would likely further compound or potentiate this. These confounding issues would play out over time, potentially outside the 28-day efficacy window, and would thus probably **be explained or recorded as unrelated to vaccination**.

1.1.8 Antigenic Imprinting Underpins COVID-19 Vaccine Failure (Part of a Trinity)

Based on the long-known (since 1960)[94] vaccine principle called “antigenic imprinting” or "original antigenic sin,”[95,96] first contact by the immune system with the SARS-COV-2 Wuhan Hu-1 vaccine strain resulted in a primary immune response to select parts of the spike protein (i.e., epitopes or antigenic domains) that generated antibodies (by B-lymphocytes) and CD4+ and CD8+ T-lymphocytes (T-cells). A fraction of these B- and T-lymphocytes then differentiated into memory B- and T-cells in the local lymph node. This **locked** future immune responses to a limited number and repertoire of antibody and Tcell responses, which could be recalled upon (re)infection and is termed **antigenic imprinting**. Consequently, when a vaccinated person was infected with a new SARS-CoV-2 variant (i.e., Alpha, Delta, Omicron, etc.), which varied from the original Wuhan Hu-1 vaccine strain in critical parts of the virus spike protein targeted by neutralizing antibodies (i.e., the receptor-binding domain, RBD), the immune system preferentially “recalled” those original Wuhan Hu-1 antibody memory responses. However, these recalled responses failed to protect against the Omicron strain because it had mutated in its RBD and other critical locations.

Studies show that Alpha, Delta, and Omicron “breakthrough infections” predominantly activated pre-existing cross-reactive memory B-cells, with only limited induction of new Omicron-specific antibody responses.[97,98,99,100,101,102] These publications confirm that antigenic imprinting plays a pivotal role in SARS-CoV-2 immunity to viral variants and helps explain why Omicron variants are vaccine escaping and why we see **Omicron vaccine failure**. Antigenic imprinting has also been demonstrated in vaccinated subjects

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boosted with Moderna's mRNA-1273 or a B.1.351/B.1.617.2 (Beta/Delta) **bivalent vaccine** (mRNA-1273.213). A bivalent booster induced a high percentage of memory B-cells (MBCs) that recognized the spike protein antigen from the original SARS-CoV-2 Wuhan Hu-1 strain. This means the MBCs generated by the primary vaccination dominated the recall response induced by the bivalent booster (preprint).[103] This bivalent vaccine finding has **important implications** for **countries with high vaccination rates** that used the original Wuhan Hu-1 strain vaccine, like New Zealand, England, Scotland, Canada, etc. and subsequently attempt to use or mandate a bi-/multi-valent vaccine as **new pandemic waves arrive**.

Antigenic imprinting is thus a **double-edged sword** because it can provide a rapid means of population protection upon (re)infection with a slightly drifted strain or be an obstacle to achieving population protection in the face of significant mutation typical of error-prone RNA viruses during pandemics. This is because antigenic imprinting comes at the expense of generating new protective immune responses against antigenically distinct epitopes resulting in **vaccine failure**.[104,105] Antigenic imprinting thus explains why zoonotic mutation-prone respiratory RNA viruses are problematic to vaccine **innovators** and global/national vaccination **strategists** during pandemics (i.e., WHO, healthcare agencies, drug regulators).

Antigenic imprinting raises **two critical issues**. Firstly, in my view, with ADE and viral mutation as a **Trinity**, it should have been a crucial part of **vaccination informed consent** (i.e., *predictable and scientifically obvious risks associated with vaccination during pandemic waves for mutation-prone viruses*). Secondly, Omicron variants could **revert to a more virulent** form (i.e., cause increased disease), which would go largely **uncontested** by an effective vaccine-induced immune response upon (re)infection, potentially even with second-generation bivalent/multivalent vaccines. Reversion to virulence was highlighted this year when a recombinant Delta-Omicron variant emerged.[106] This publication states, "*This recombinant exhibits immune escape properties similar to Omicron, while its behavior in mice expressing the human ACE2 receptor is more similar to Delta*" (i.e., the data showed it was more pathogenic).[107]

1.1.9 Did ADE, Antigenic Imprinting, and Viral Mutation Risks Versus High Influenza-like Survival Rates in Sub-70 year Demographics and Superior Natural Immunity Support Whole Population Vaccination?

Meta-analysis studies covering the 2020 phase of the pandemic confirmed a median infection fatality rate of 0.15%[108] to 0.27%, which reduced to a **median of 0.05%** for people younger than 70 years of age (i.e., 50 per 100,000 infected).[109] This means those over 70 years of age carried the burden of COVID-19 disease and death and were at the most risk of severe disease. By comparison, the estimated global mortality rate for **seasonal influenza was 0.04%**, similar to COVID-19 for those under 70 years of age.[110]

Two global reviews covering 2020, the worst part of the pandemic, revealed **high survival rates** in healthy

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adults, youth, and kids (*Study-1:*[111] *0-19yr: 99.9973%, 20-29yr: 99.986%, 30-39yr: 99.969%. 40-49yr: 99.918%. Study-2:*[112] *0-34yrs: 99.996%. 35-44yrs: 99.932%*). For risk calibration, SARS-CoV-2 fatality rates for 0-34yr age groups were on par with automobile and other accident fatalities (Study-2). Furthermore, mortality rates for those younger than 18 years old were less than 0.003%, or 3 per 100,000, **comparable to influenza** (CDC, Table 2).[113] In my view, survival rates for kids,[114] youth, and working-age adults were higher than government healthcare and media narratives suggested.

Omicron's disease severity was **significantly lower** than for Delta (i.e., >50-70%, $p < .05$ for *progression to symptomatic disease, hospital admission, ICU admission, mechanical ventilation, length of stay, and death*) but was associated with much higher transmissibility than earlier SARS-CoV-2 variants.[115,116,117,118,119,120,121,122,123] The COVID-19 pandemic from the Wuhan Hu-1 to the Omicron appeared to follow a trend from higher virulence and lower transmissibility in the first pandemic wave to higher levels of transmission with **significantly lower virulence** in the Omicron wave (*see OWID graphic, global.*[124] *UKHSA Figures 53, 58 and pgs.65, 75,*[125] *Scotland data: Figures 7 and 11.*[126]). This suggests transmissibility was a Darwinian trait during this pandemic, or rather sick-moribund people don't transmit the virus as well as asymptomatic or mildly ill and publicly circulating people. This appears similar to my observed trends across 500 years of influenza pandemics (i.e., private research, **see hyperlink**).[127]

In my view, by **conflating** high transmissibility with high virulence (esp. Omicron) and using this to motivate vaccination in all population demographics, the lower-risk part of humanity was **deprived of the opportunity** to develop natural immunity, which is superior to COVID-19 vaccination (i.e., *duration of protection, cross-protection, broader antiviral T-cell and B-cell immunity*). See 200+ publications via 2-citation links to comprehend this statement of opinion.[128,129] Governments also had non-vaccine options for prophylactic disease management (i.e., Ivermectin) without vaccinating the whole population (section 2.6).

1.2 Evidence of Toxic COVID-19 Vaccine Lots Under FDA Jurisdiction Had Global Implications

The bottom line: According to my analysis of the US Government's Vaccine Adverse Event Reporting System data (VAERS),[130] one year of COVID-19 vaccine-associated deaths and hospitalizations ("adverse outcomes", by 07/12/2021) were equivalent in number to **all other vaccine adverse outcomes** in the USA over the last **32 and 20 years** respectively. A small minority of vaccine lots was associated with the majority of these COVID-19 vaccine-related adverse outcomes. Furthermore, there was an uneven distribution of adverse outcomes across vaccine lots (i.e., **skewed and peaked**). Most of these adverse outcomes were associated with a minority of lots sent to a **larger number of states**. This minority of lots had a significantly higher weighted mean and median of adverse outcomes per state per lot fraction shipped to a state when

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822 lots were sent to ≥11 states (deaths) and ≥19 states (hospitalizations) compared with those sent to state
823 totals below these thresholds. These issues were replicated with all US COVID-19 vaccines. These results
824 would imply the presence of significant differences in vaccine lot composition or **specification,** or the
825 targeted vaccine use in high-risk demographics (i.e., the elderly) coordinated via a **central vaccine**
826 **distribution mechanism**. Ninety percent of all vaccine-related adverse outcomes were associated with
827 mRNA gene-therapy-vaccines.

828 There were 20,556 lots associated with unique lot numbers and adverse outcomes after one year of
829 population-level vaccine use in the USA. COVID-19 vaccine-related deaths were equivalent to 32 years of
830 all vaccine-related deaths in the USA, comprising 15.4yrs for BNT162b2 (Comirnaty), 13.2yrs for Spikevax,
831 and 3.2yrs for Janssen's COVID-19 vaccine. COVID-19 vaccine-related hospitalizations were equivalent
832 to 20 years of all vaccine-related hospitalizations in the USA, comprising 10.5 years for Comirnaty, 7.6
833 years for Spikevax, and 3.2 years for Janssen's COVID-19 vaccine.

834 There were 10,428 deaths, of which 7,259 were associated with 775 lots identified by a lot number. This
835 yielded a mean of 9.4 (95% confidence interval 8.9-9.8, minimum 1, maximum 142) and a median of 1.0
836 death per lot. The number of lot deaths demonstrated skewness and peakness, indicating an uneven
837 distribution among lots. Fifty-seven and 118 lots identified by lot numbers accounted for half and three-
838 quarters of all COVID-19 vaccine-associated deaths, respectively. A minority of COVID-19 vaccine lots
839 sent to 11 or more states (n = 123 of 775) accounted for 75% of all deaths and were associated with a
840 weighted- mean of 2.54 and a median of 2.36 deaths per state per lot fraction sent to a state. By contrast,
841 those lots sent to 10 or fewer states were associated with a weighted- mean of 1.30 and a median of 1.00
842 deaths per state per lot fraction sent to a state. These weighted mean and median-shape differences were
843 statistically significant (Welch's unpaired T-test and Mann-Whitney U-test, respectively, all $p < .0001$).

844 There were 48,851 hospitalizations, of which 33,632 were associated with 2,508 lots identified with a lot
845 number. This yielded a mean of 13.4 (95% confidence interval 12.9-13.9, minimum 1, maximum 489) and
846 a median of 1.0 hospitalizations per lot. The number of lot hospitalizations demonstrated skewness and
847 peakness, indicating an uneven distribution among lots. Eighty-four and 165 lots identified with lot numbers
848 accounted for half and three-quarters of all COVID-19 vaccine-associated hospitalizations, respectively. A
849 minority of COVID-19 vaccine lots sent to 19 or more states (n = 203 of 2,508) accounted for 84% of all
850 hospitalizations and were associated with a weighted- mean of 4.66 and median of 4.31 hospitalizations per
851 state per lot fraction sent to a state. By contrast, those lots sent to 18 or fewer states were associated with a
852 weighted- mean of 1.42 and a median of 1.00 hospitalizations per state per lot fraction sent to a state. These
853 weighted mean and median-shape differences were statistically significant (Welch's unpaired T-test and
854 Mann-Whitney U-test, respectively, all $p < .0001$).

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A Chi-square goodness-of-fit test demonstrated the observed distribution of total COVID-19 vaccine-related deaths and hospitalizations when grouped by ≥11 states and ≤10 states (deaths), and ≥19 states and ≤18 states (hospitalizations) differed significantly from their expected distributions (all $p < .00001$). The expected total lot-associated adverse outcomes were derived by proportioning the observed lot-associated adverse outcomes according to the totals (i.e., by groupings ≥11 or ≤10 for deaths and ≥19 or ≤18 states for hospitalizations) and the total number of states lots were sent to.

This analysis was done with an appreciation that VAERS data must be interpreted with caution, given the inherent limitations of passive pharmacovigilance surveillance systems. The primary use of the VAERS, therefore, should be for early safety signal detection and directing regulatory and medical research investigation. This explains why I confined my analysis to serious adverse outcomes associated with **lot numbers**, thus providing a direct association with the COVID-19 gene-therapy-vaccine.[131] In reflecting on the issues mentioned above, the combination of an unprecedented level of COVID-19 vaccine-associated deaths and hospitalizations, a skewness-peakness of adverse outcomes across vaccine lots, and a US State clustering pattern of adverse outcomes all linked to lot numbers should have alerted the FDA and Center for Disease Control (CDC) there was a **safety problem in need of investigation**. In my view, that investigation should have occurred before approving expanded vaccine use in young children, youth, and pregnant women, for adult boosters, and before enforcing Government mandates on employees. Did the FDA and CDC meet their **commitment to review** the VAERS and other safety data by utilizing statistical data-mining methods to detect safety signals via a weekly and bi-weekly process (pg.6)?[132]

It is extremely concerning that after one year of COVID-19 vaccine use and unprecedented levels of serious adverse outcomes the observed level of serious or severe adverse events (SAE) was potentially **highly under-reported.** Independent VAERS research showed the observed SAE relative to expected SAE based on the Comirnaty Phase 3 clinical study SAE rate and vaccine doses administered showed an **Under Reporting Factor of 31 times** (data to 06/08/2021).[133] Furthermore, this prior cited VAERS research provided evidence for **deleted and delayed entry** of reports and the **re-coding** from **severe to mild**, further exaggerating the under-reporting. Historically, healthcare professionals, for which VAERS reporting is mandatory, and vaccine companies comprised the majority of these VAERS submissions (68%), meaning the VAERS data has some **validity** in highlighting safety signals worthy of regulatory and medical hypothesis-driven investigation.[134] Was this done?

To give a safety perspective to the above data regarding **market withdrawals on safety grounds,** three vaccine withdrawals occurred in the USA between 1976 and 2019: one for swine flu (53 deaths, Guillain Barre Syndrome one case per 100,000 vaccinated, 45 million vaccinated),[135] Rotashield (15 cases of intussusception, 1 case per 10,000 vaccinated),[136] and Nasalflu (Bell's Palsy, 13 excess cases per 10,000

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vaccinated).[137] That was the **old normal**. Giving further perspective for a high market volume seasonal flu vaccine used each year (i.e., 2018-19: 169 million doses were distributed, but not all got utilized),[138] COVID-19 vaccine-related deaths were 10x, and hospitalizations 5.4x all seasonal influenza vaccine-related deaths and hospitalizations respectively in the USA since 1990. According to the web archive, 632 million doses had been administered in the first year of its launch.[139] As of 01/12/2022, the VAERS database showed 18,557 deaths and 89,085 hospitalizations associated with COVID-19 vaccination in the USA since EUA approval, representing a 78% and 82% increase respectively since my last VAERS data download on 07/12/2021.[140] When will all COVID-19 vaccines be **withdrawn from the market**?

1.2.1 Vaccine Development Experts Identified Vaccine Safety Signals from VAERS Data

A detailed analysis of the VAERS data was also undertaken by several vaccine industry professionals at "How Bad is my Batch" ("HBIMB team"),[141] including Dr. Mike Yeadon (*former Vice President & Chief Scientific Officer of Allergy & Respiratory at Pfizer Global R&D*).[142]

Based on their analysis, the following strong VAERS safety signals were identified:

1) Toxic lots were part of a mathematical series of lot numbers: for example, Pfizer lots with the same first two letters (i.e., EN, EP, ER, EW, etc.) tended to occupy distinct ranges of adverse outcomes, with toxicity decreasing as the alphabet ascended. Within each alphabetical group, there were some high-toxicity lots and a larger number of low-toxicity lots, with little in between (i.e., a sudden drop from the 2000 range to 37). If adverse outcomes were a random result of individual comorbidities, then why were they predominantly occurring with vaccine lots that were *part of a mathematical-alphabetical series* (i.e., EN6198, EN6199, EN6200, EN6201, EN6202, EN6203, EN6204, EN6205, EN6206, EN6207, EN6208, or EW0150 to EW0217 for almost all deaths and disabilities in children)? Statisticians concluded that this safety signal was **non-random**.[143]
2) Percent mRNA stability: this explained one-third and half of the lot variability in deaths and serious adverse events, respectively, with a higher percentage of mRNA stability associated with a higher rate of adverse outcomes indicating the **biologically active non-degraded mRNA was toxic-harmful**.[144]
3) US State bias: some states like Kentucky, Montana, Alaska, Tennessee, and North and South Dakota experienced 4x-11x the number of deaths per 100,000 vaccinated, suggesting they received more toxic batches or these were administered to more vulnerable people.[145]
4) Statistical clustering around the vaccination date: a high proportion of deaths occurred on the vaccination day, with many people dying within 2 hours of vaccination. VAERS data and Pfizer's post-EUA 90-day adverse event report submitted to the FDA confirmed that most deaths occurred within 24 hours of vaccination.[146,147] Seventy percent of all individuals experiencing adverse events had an onset of symptoms within 48 hours following the first or second doses (Chi-square statistic, $p < .0001$).[148]

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5) Age bias: age explained one-third of the lot variability in deaths. Approximately three-quarters were older than 60yrs, and one-quarter were aged 40-60yrs. COVID-19 vaccines tended to afflict the old with death and the younger age groups with severe injury or chronic illness.[149]
6) Gender bias: women experienced far more adverse effects than men, yet this critical safety information is missing from their **informed consent worldwide**.[150,151]

The HBIMB team reviewed various European Medicines Agency (EMA), FDA, and Pfizer documents supporting Comirnaty's EUA approval and discovered that Pfizer utilized two different non-cGMP-compliant manufacturing processes to support its FDA EUA approval.[152,153,154] The mRNA drug substance was also *highly unstable* and was unprecedentedly permitted to contain up to 50% degraded mRNA fragments. According to the HBIMB team's research, these mRNA degradants had not been characterized and their biological activity was unknown. The final dose vials were also not characterized given technical issues associated with creating a well-mixed, homogenous, and consistent final dose form linked to mRNA fragility. This meant the active mRNA ingredient was unevenly distributed among lot vials, resulting in more and less toxic vials within the same lot, while the mRNA was then rapidly degraded.

Batches with a higher percentage of intact mRNA were **significantly more toxic**, and the relative toxicity (i.e., *percentage of serious adverse events-to-total adverse events*) dropped off rapidly in the first 30-40 days post-manufacture before plateauing. Their regression modeling showed that more than half of the lot variability in toxicity was explained by the percentage of intact mRNA (r-squared = 0.56). Therefore, factors associated with a high percentage of intact mRNA, which putatively enhanced serious adverse outcomes, included proximity to the manufacturing date, shorter transit-storage times from the manufacturing site to the final place-time of use, and high demand and thus a shorter duration of storage (i.e., *created by vaccine mandates and employer policies "jabbed for jobs", conflating Omicron's high-transmissibility with high virulence in media, etc.*).

1.2.2 Comirnaty is Composed of Toxic and Hazardous Chemicals Whose Toxicological Properties were Not Fully Investigated (Safety Data Sheet Disclosures)

The question is if the non-mRNA ingredient specifications of Comirnaty were exceeded, or it contained unknown toxic substance(s), then what could theoretically be implicated?

Pfizer's Safety Data Sheet (SDS) confirms Comirnaty is synonymous with BNT162b2, which is synonymous with PF-07302048 (i.e., the compound number), all containing PF-07305885. As a chemical family Comirnaty is described as "Lipid Nanoparticles containing PF-07305885 (BNT162b2) and Lipids".[155] Two issues arise from the Comirnaty SDS that raised questions about its composition. Firstly, why is PF-07305885 listed as an **undisclosed proprietary chemical** in the SDS but is not evident in any

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FDA or European Medicines Agency (EMA) regulatory documents? The Comirnaty compound number is PF-07302048, as was confirmed in FDA and EMA Documents.[156,157] Compound PF-07302048 is distinct from PF-07305885, as detailed in the SDS section 2.2 ("Mixtures"). Compare the product contents between the cited and SDS and Table P.1-1 of the following cited document.[158] **What is PF-07305885?**

The SDS section 5.2 ("Specific hazards arising from the chemical") indicate Comirnaty was nominally classified as a "Chemical" or "**Hazardous Substance**" as interpreted under the HSNO Act 1996 (SDS section 2).[159] Section 3.2 of the SDS lists two chemicals in the lipid nanoparticle formulation encapsulating the mRNA, **ALC-0315** (cationic lipid) and **ALC-0159** (PEG-lipid). The EMA describes these chemicals as "novel" and confirms **complete information was not provided**,[160] which is similarly described in the FDA document.[161] Safety data sheets for non-Comirnaty research grade versions of ALC-0159[162] and ALC-0315[163] highlight significant **safety and toxicity issues**, including heart and liver damage, CNS depression, anemia, headache, lassitude, drowsiness, narcosis, cough, reproductive and teratogenic effects. SDS section 11.1 ("Information on hazard classes as defined in EC Regulation No. 1272/2008") warns that toxicological properties were **not fully investigated** before its approval.

Comirnaty's SDS highlights that local and systemic side effects may occur during the **accidental injection**. The SDS section 7.1 ("Precautions for safe handling") stipulates exposure to this hazardous chemical mixture via inhalation, and contact with the skin, eyes, and clothing should be avoided. The toxicity of Comirnaty is indicated in SDS section 4.1, which describes **first aid measures** for inhalation, eye and skin contact, and ingestion, and instructs people to seek medical attention. Section 8 advises using appropriate **personal protective equipment** when handling this hazardous chemical mixture, including wearing impervious gloves and disposable clothing and full body protection when handling Comirnaty to prevent skin contact. Section 5.3 ("Special protective equipment for fire-fighters") indicates potential harm by advising firefighters to wear self-contained breathing apparatus, full firefighting gear, and personal protection equipment. These extreme exposure protection measures **discourage the notion that Comirnaty is safe**.

1.3 Pathogenesis Mechanisms Underpinning Vaccine-Associated Enhanced Disease (Spike Protein & Lipid Nanoparticle Related)

This section reviews the predictable pathogenesis mechanisms associated with mRNA lipid nanoparticle-based COVID-19 vaccines and is focused on Comirnaty for reasons previously stated, as well as through Pfizer's 90-day Cumulative Analysis of Post-authorization Adverse Event Report (i.e., a **Pfizer FOI disclosure**). **At least five broad pathogenesis mechanisms** exist by which virus-free spike proteins can directly cause disease or exacerbate **preexisting comorbidities** common to severe COVID-19 outcomes**,**

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in addition to lipid nanoparticle proinflammatory reactogenicity and complement activation-related pseudo-allergy (CARPA, section 1.3.2). These pathogenesis mechanisms include angiotensin-converting enzyme-2 receptor (ACE2) and other ligand interactions (CD147), exosomes, immune-mediated/autoimmunity (section 1.3.3), prion diseases (section 1.3.4), and ADE/antigenic imprinting (section 1.1). This mechanistic organization also provides a lens to understand the **strategic intentions of coronavirus gain-of-function strategists** since SARS-CoV-1 (2002, section 2). As a former vaccine innovator of computationally designed synthetic long-peptide-based vaccines targeting high-mutation-prone RNA viruses that cause zoonoses-pandemics and infect a genetically diverse human population, I wanted to understand what pathogenic mechanisms were inserted or relied upon at the **point of innovation** in the minds of "the" gain-of-function **strategists** and those having an influence on global vaccination strategies.

1.3.1 Pfizer was Unprepared for the Sheer Volume of Comirnaty Adverse Event Reports, which Revealed Predictable Safety Signals

Preamble: Given the specific purpose underpinning my VAERS lot numbered death and hospitalization analysis (section 1.2, i.e., *to identify statistical evidence for toxic COVID-19 vaccine lots*) and the large volume of associated adverse events (699,839), deaths (10,428), and hospitalizations (48,851) I elected not to review their associated symptoms, diseases, and medical conditions. For this purpose, I reviewed Pfizer's 90-day cumulative analysis of post-authorization adverse event (AE) reports for Comirnaty submitted to the FDA on 28/02/2021 (FOI),[164] in which the USA accounted for one-third of the case reports. These case reports were processed by pharmacovigilance professionals, which adds a degree of credibility to any analysis of symptoms, diseases, and medical conditions used to define harm and risk factors.

Pfizer confirmed its **unpreparedness for this sheer volume** of AEs by stating, "*due to the large numbers of spontaneous adverse event reports received for the product, the marketing authorization holder has prioritized the processing of serious cases*." This statement implies caution is merited in any interpretation or analysis because it indicates a **bias** could have been introduced in what and how much data was provided in their report. Pfizer was forced to upgrade its supporting technology, implement process solutions, and significantly increase its headcount to deal with this unprecedented volume of AE reports (pg.6).

Pfizer reported 42,086 adverse event cases, which resulted in 1,223 **deaths** (2.9%), and 11,361 **unrecovered** AE cases (27%). There were 158,893 adverse events, with 3.8 AEs per case, which complements observations that 95% of people who died from COVID-19 had an average of four comorbidities (section 1.3.3).[165] Approximately one-third of case reports were accounted for by the USA and UK each, 18% by five EU countries (i.e., 82% by NATO nations), with the balance spread over 56 countries. Seven system organ classifications accounted for 82% of AEs. One-third of AEs were categorized as general and injection site related (i.e., local and systemic reactogenicity), 16.3% nervous

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system disorders, 10.9% musculoskeletal and connective tissue disorders, 8.9% gastrointestinal disorders, 5.6% respiratory and chest cavity disorders, 5.3% skin and subcutaneous disorders, 2.9% Infections.

Pfizer utilized a list of 1,290 AEs of special interest to interrogate the case report data (Appendix 1, pgs. 30-38). This list appears to comprise serious/severe AEs identified in the Phase-3 study, the list of 30 AEs compiled by the FDA before EUA approval (pg.17),[166] and historical vaccine AEs (general), among others. **Safety signals emerge** by **(re)grouping** Comirnaty's adverse events into broad pathogenesis mechanism categories associated with virus-free spike proteins (section 1.3.3), **(1)** immunization effects (i.e., ADE, antigenic imprinting), **(2)** vaccine-associated enhanced disease (VAED) associated with tissues-organs rich in **ACE2** and **CD147** receptors and exosomes, and **(3)** lipid nanoparticle proinflammatory reactogenicity and CARPA/anaphylaxis, and **(4)** immune-mediated/autoimmunity. These pathology mechanisms also find support in various safety reviews.[167,168,169] **Pfizer listed** this same array of implied pathogenesis mechanisms via its categorization of diseases, organs and tissues, and symptoms as adverse events of special interest or used them in its VAED search criteria (Tables 5 **footnote a**, and 7), including vascular endothelium- and blood clotting-related, and heart, respiratory, brain, kidney, and gastrointestinal organs.

Pfizer identified VAED as a significant identified and potential risk. Surprisingly, there was **no mention of ADE** in their list of reportable risks. Considering the wealth of spike protein antigen-based vaccine prototype literature on ADE and its associated disease severity and mortality outcomes in animal studies, I would have expected ADE to be **monitored alongside** VAED. There were 138 VAED cases, mainly serious, reporting 317 events of suspected VAED, including its respiratory variant, of which 38 died. There were also 1,927 COVID-19 infections confirmed among the vaccinated (i.e., 4.6% of cases). I believe these apparent COVID-19 vaccine failures would place **ADE and antigenic imprinting** at the top of the list of differential diagnoses (i.e., medical possibilities). In my view, **ADE should have been listed alongside VAED** in Pfizer's pharmacovigilance plan and then been actively monitored in post-marketing studies insisted upon by regulators and healthcare agencies (i.e., **the old normal**).

By grouping **local and systemic reactogenicity** AEs together, more than half of the 93,473 AEs reported in ≥2% of cases could potentially have been associated with a robust pro-inflammatory response induced by the LNPs (Table 2, pgs.8-9). Furthermore, and potentially related, there were 2,958 relevant anaphylaxis AEs, of which 2,341 were serious, and nine died. This corresponded with 1,002 cases meeting specified criteria (2.4%). Anaphylaxis median onset latencies were within hours (Table 4, page 10). This Pfizer report did not discuss **complement activation-related pseudo-allergy (CARPA**) in its review of anaphylaxis. CARPA is a potentially lethal anaphylatoxin/mast-cell mediated systemic-circulatory-stress response to chemical toxicity (i.e., PEG-lipid associated, section 1.3.2).

By grouping cardiac, gastrointestinal, and nervous system disorders together under the assumption that

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ACE2 receptors were highly expressed in those tissues (section 1.4.5), 27% of 93,473 AEs could potentially be associated with ACE2-spike protein-associated pathologies. On an AE of special interest basis, 8.3% of 42,086 adverse event cases (i.e., with an average of 3.8 AEs per case) had pathologies associated with **ACE2 and its overlapping distribution with CD147 receptor-expressing and exosome-associated tissues and organs** (i.e., *cardio-, cerebro-, pulmonary-, and renal- vascular endothelium, and heart, brain, lung, and kidney, Table 7, pgs.16-24, section 1.3.3*). Median onset latencies were <24 hours for cardiovascular, one day for hematological and neurological, and four days for renal AEs.

Immune-mediated and autoimmune AEs of special interest (AESI) represented 2.5% of cases (1,050), resulting in 780 serious and 12 fatal AEs affecting the central and peripheral nervous system, heart, skin, and pancreas. Musculoskeletal AESIs comprised 8.5% of cases resulting in 3,640 AEs, of which 1,614 were serious. Arthralgia accounted for the majority (3,525), with arthritis, rheumatoid arthritis, polyneuropathy, and post-viral fatigue syndrome the balance (Table 7, pg.20). Arthralgia may be linked to autoimmunity,[170] which could implicate spike protein mimicry/cross-reactivity (T- and/or B-cell) and/or LNP formulation- and mRNA innate immunity- induced proinflammatory responses (section 1.3.2). Median onset latencies were <24 hours for immune-mediated/autoimmune and one day for musculoskeletal AEs (section 1.3.3).

Given the limitations of spontaneously reported pharmacovigilance data, the unavailability of data for the number of vaccine doses administered alongside these AE case reports, and the absence of COVID-19 disease rate data for this specific period of the pandemic, it is my opinion that Pfizer's conclusion regarding Comirnaty's "*favorable benefit: risk balance*" was **unsubstantiated** by any quantitative results of a risk-to-benefit analysis provided in their report. In each listed AESI, Pfizer concluded, "*this cumulative case review does not raise new safety issues.*" In my opinion, Pfizer's 90-day post-EUA safety assessment fell short of its potential, and our safety understanding at EUA was compromised by **regulators failing to demand** pertinent preclinical and clinical safety information before EUA approval (sections 1.4-5).

These predictable pathologies help explain the **burgeoning list of scientific publications** on COVID-19 vaccine harm and lethality (*1,250 safety-related publications: generally,* [171] *children* [172]) and the **unprecedented increase** in health and life **insurance claims and payouts** in 2021-2022 as an insurance industry-wide phenomenon on more than one continent.[173,174,175,176,177,178,179,180]

1.3.2 Lipid Nanoparticles (LNPs) are Pro-Inflammatory and Toxic

Under the presumption that the mRNA-LNP technology platforms used by mRNA gene-therapy-vaccines were non-inflammatory, this would explain why local and systemic reactogenicity were conflated with robust immune responses generated by the mRNA vaccines in commentaries (in general). This reactogenicity could represent a strong innate inflammatory response induced by the LNP formulation

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chemicals (i.e., inflammatory cytokines and chemokines, including thousands of upregulated genes).[181] The pre-COVID-19 literature also detailed the proinflammatory nature of mRNA-LNPs, which was predominantly associated with the LNP formulation used to encase the mRNA. This pro-inflammatory feature was consistent across multiple species of animals, while chronic dosing with mRNA-LNPs produced toxic side effects, including liver damage.[182,183]

The Comirnaty SDS specifies allergic reactions, including anaphylaxis, that may occur with accidental injection. This is because the PEG-lipid (ALC-0159) essentially exchanges out of the lipid nanoparticles before cellular uptake (pg.53),[184] making this PEG-lipid more bioavailable. This enhanced bioavailability could be implicated in the so-called COVID-19 mRNA gene-therapy-vaccine anaphylaxis problem seen with COVID-19 gene-therapy-vaccines,[185,186,187,188,189] including Comirnaty.[190,191,192] Chronic dosing studies with mRNA-LNPs describe anaphylaxis as complement activation-related pseudo-allergy (CARPA), which is a potentially lethal anaphylatoxin/mast-cell mediated systemic-circulatory-stress response to chemical toxicity (i.e., polyethylene glycol, PEG).[193,194]

Thus, with drug **regulators failing to demand** toxicology data for Comirnaty's LNP formulation chemicals before EUA (section 1.4), including inflammatory cytokines and chemokines, the ability to identify the specific cause of the local and systemic reactogenicity and anaphylaxis/CARPA safety problems, and discern potent immune responses from pro-inflammatory augmented immune responses were **eliminated**.

1.3.3 Vaccine-Induced Spike Proteins Drive an Array of Pathogenesis Mechanisms that Trigger Pathologies and Exacerbate Comorbidities

This section reviews **three broad pathology mechanisms** by which SARS-CoV-2 and virus-free spike proteins can directly cause or exacerbate comorbid diseases. Furin, a cell surface protease (i.e., enzyme protein scissors), is a common denominator linking SARS-CoV-2 spike protein binding to the ACE2 receptor (i.e., infectivity, pathogenicity),[195,196,197,198] in **ACE2-rich tissues and organs**, which overlaps with the most prevalent **comorbidities** involving tissues and organs associated with severe COVID-19 outcomes, in at-risk populations (i.e., elderly, males) (see below). These mechanisms **place furin**, common preexisting or **comorbid diseases** in the elderly at risk, and SARS-CoV-2's uniquely encoded **furin cleavage site** (FCS) **center stage** (section 2, gain-of-function). This unique FCS is also part of SARS-CoV-2's nuclear localization signal sequence in what appears to be a 2-in-1 genetic insertion aimed at enhancing infectivity and pathogenicity in humans (section 2.2.1, gain-of-function).

Virus-free spike proteins: The vaccine mRNA-manufactured spike proteins peak in human plasma within five days, circulate in the plasma for weeks after the first vaccination,[199] and are detectable in lymph nodes two months after vaccination.[200] In addition, vaccine-delivered mRNA moves rapidly from the injection

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site throughout the body of animals, peaks within 6-48 hours, and accumulates in the heart, lungs, brain, liver, lymph nodes, spleen, adrenal glands, gonads, among other tissues.[201,202,203,204] Therefore, virus-free spike proteins arise from mRNA transcription at the **injection site** and putatively by **tissues up taking the mRNA** distant from the injection site.

Mechanism 1: Virus-free spike proteins. During SARS-CoV-1 and SARS-CoV-2 infections, the spike protein receptor-binding domain (RBD) binds to the human ACE2, triggering viral entry and pathogenesis.[205,206,207] The ACE2 receptors predominate in lung alveoli and respiratory surfaces, blood vessel linings (i.e., endothelium), heart muscle, arterial smooth muscle, brain, intestines, kidney, skin, lymphoid system, hematopoietic stem cells, endocrine, and reproductive tissues.[208,209,210,211,212,213] Furthermore, increased ACE2 expression occurs in heart disease, hypertension, and dementia,[214,215] putatively indicating an enhanced disease susceptibility to SARS-CoV-2 infection or vaccine-induced spike proteins. The most prevalent **comorbidities** and most significant risk factors associated with severe COVID-19 outcomes are associated with the cardiovascular system (incl. hypertension, cardiac arrhythmias), chronic obstructive pulmonary disease, obesity, diabetes mellitus, cancer, cerebrovascular accidents, dementia, and acute and chronic kidney disease.[216,217,218,219,220,221,222] This range of comorbidities may reflect SARS-CoV-2's **tissue tropism** for vascular endothelium, the cardiovascular system, respiratory tract, brain, and kidney.[223] Older age and male gender were also risk factors for severe COVID-19 outcomes.[224,225,226]

The SARS-CoV-2 spike protein and its S1 sub-unit, **free of the virus,** are capable of causing human vascular **endothelial** damage and dysfunction in a dose- and time-dependent manner.[227,228] This puts the spotlight on the time-limited vaccine production of spike proteins by humans and their release into the blood circulation (or via exosomes). Virus-free spike protein induces degradation of **brain** endothelial junctional proteins,[229] and dysregulates the vascular and immune functions of brain pericytes by triggering cellular stress,[230] resulting in a pro-inflammatory response and alterations to the blood-brain barrier function.[231] The expression of ACE2 in brain vascular pericytes and endothelial cells is modulated by spike protein in a dose- and flow-dependent manner.[232,233] Virus-free spike protein binds to **ACE2 receptors** and causes ACE2 downregulation, which inhibits mitochondrial function,[234] causes oxidative stress and inflammation and triggers **blood clotting mechanisms**.[235,236,237] This milieu of damage contributes to the severity of **lung** pathologies[238] and predisposes to **myocardial infarction, stroke**, and **renal** injury.[239,240]

Virus-free spike proteins bind **the CD147 receptor**, mainly expressed in the **heart, kidneys, and lungs**. Activation of CD147 by spike protein promotes cardiac hypertrophy and failure[241] and **triggers** microvascular injury, inflammation, and blood clotting mechanisms via cardiac pericytes.[242] Vascular injury may also be mediated by heat shock protein 90,[243] and androgen (i.e., male), TNF-α, and **other**

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signaling pathways.[244,245] The spike protein also promotes ACE2-independent vascular endothelium growth factor upregulation in animal enterocytes leading to **intestinal** inflammation.[246]

Mechanism 2: Spike protein exosomes. Exosomes circulate throughout the body after infection and vaccination. Exosomes are cell-secreted microvesicles that arise for physiological and pathological reasons, including in response to microbial attack and stress conditions.[247,248,249,250,251] In general, exosomes are involved in various disease processes, including inflammation, oxidative stress, endothelial dysfunction, thrombosis, hemostasis, cardiovascular disease, and cardiac dysfunction.[252,253,254,255] Thus, it is unsurprising that SARS-CoV-2 exosomes have been implicated in **inflammation, coagulation, complement pathways, and immunomodulation**, with exosome-associated biomarkers correlating with disease severity.[256] Comirnaty vaccination also induced exosomes containing the spike protein S2 sub-unit, which were detectable in plasma **14 days after the first vaccination**, were significantly boosted by 14 days after the second dose and were still detectable **four months later**. Spike protein-loaded exosome kinetics tracked the antibody response indicating a potential role in immunogenicity as well.[257,258]

Mechanism 3: Autoimmunity and immune-mediated. COVID-19 vaccination with mRNA, viral vectors, and inactivated vaccines[259,260] have been associated with new-onset and flare-ups of autoimmune disease including autoimmune hepatitis,[261,262,263] hematologic autoimmunities,[264,265] Guillain-Barré syndrome,[266] IgA nephropathy, CNS demyelination autoimmunities,[267] encephalitis autoimmunities,[268] among others. However, information regarding the risk of vaccine-associated autoimmune disease is controversial and is hindered by the low incidence and the **diverse array** of autoimmune diseases.[269] The vaccine risk of autoimmunity prioritizes knowing which tissues **uptake the spike protein mRNA** and the **tissue-organ sensitivity** to pro-inflammatory lipid nanoparticles (sections 1.4.1-3, preclinical safety study deficits).

The main mechanisms by which COVID-19 vaccines putatively trigger autoimmunity include molecular mimicry and cross-reactivity resulting in auto-antibody and auto-Tcell mediated self-attack, proinflammatory vaccine adjuvants and immunostimulants that help break self-tolerance, and non-specific bystander activation.[270,271,272] Researchers demonstrated a high degree of proven and predicted mimicry and cross-reactivity between the SARS-CoV-2 spike protein and human tissue antigens, mediated by T- and B-cells.[273] This cross-reactivity included human *barrier proteins, lung surfactant, cardiovascular, lung, nervous system, gastrointestinal, connective tissues, and thyroid tissues, among others.*[274,275,276,277,278,279] As such, molecular mimicry and cross-reactivity mediated by T-cells and auto-antibodies could play a role in the multi-system disease processes of COVID-19 infection and vaccination. The mRNA in the COVID-19 vaccine also acts as an immunostimulant engaging Toll-like receptors and intracellular inflammasome components to trigger inflammation and immunity,[280] as do lipid nanoparticle formulations.[281]

Cells in tissues up taking the gene-therapy-vaccine mRNA, which then transcribes that into spike proteins

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and present CD8+ Tcell epitopes on their cell surface in HLA class I molecules (i.e., **non-self**, **self/mimicry**),[282,283] and potentially non-traditional antigen-presenting cells expressing CD4+ Tcell epitopes in HLA class II molecules (i.e., respiratory and gastrointestinal tracts),[284] could also then become the target of non-self- and self- immune-mediated attack and autoimmunity.[285] This Tcell epitope presentation is a normal friend-or-foe immunological surveillance process operating via the human leukocyte antigen system (HLA).

Conclusion: Given these predictable pathogenesis mechanisms by which virus-free spike proteins can cause disease or exacerbate preexisting **comorbid diseases** common to severe COVID-19 outcomes (i.e., **shared tissue-organ tropism-distribution; furin and ACE2**) it was interesting to observe that **Pfizer listed** this same array of diseases, organs and tissues, and symptoms as adverse events of special interest or used them in its search criteria for vaccine-associated enhanced disease (Tables 5 **footnote a**, and 7). These included vascular endothelium and blood clotting-related, and heart, respiratory, brain, kidney, and gastrointestinal organs.[286]

1.3.4 Spike Protein Inducible Prion Diseases are a Potential Ticking Time Bomb

The SARS-CoV-2 Wuhan Hu-1 spike protein encoded by the Comirnaty and Spikevax mRNA has several features that could pose a **prion disease risk**. Prions represent misfolded proteins that can self-propagate and cause **neurodegenerative diseases** due to the formation of toxic protein aggregates in the brain. Prion diseases like amyotrophic lateral sclerosis, frontotemporal lobar degeneration, Alzheimer's, and Huntington's disease are usually rapidly progressive and always fatal.[287,288]

The Wuhan Hu-1 spike protein possesses several heparin-binding sites within the spike protein S1 subunit, which bind heparin and other aggregation-prone heparin-binding proteins.[289,290] A **prion-like domain** (PrD) resides within the receptor-binding domain (RBD), and five of the seven amino acids that contact the RBD with the ACE2 receptor are located within this PrD, which is thought to facilitate viral adhesion and cell entry.[291] Theoretically, the conformationally altered spike protein RBD (i.e., prefusion stabilized, 1-up-2-down configuration) could seed-catalyze the aggregation of brain aggregation-prone proteins (i.e., *beta-amyloid, α-synuclein, tau, TDP-43*.), which are at high levels in the brain, and are known to be associated with neurodegenerative diseases.[292,293]

The spike protein also contains five prion sequences comprising two glycine amino acids spaced by three amino acids, termed a **glycine zipper motif (GxxxG)**, which has been linked to protein misfolding susceptibility. Thus, it is plausible the zipper motifs contained in freely circulating- or exosome-containing-spike proteins after COVID-19 vaccination and/or associated with post-vaccination COVID-19 infection (i.e., enhanced risk) could behave as a prion and be associated with neurodegenerative diseases in the future.

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Giving context to the spike protein zipper motifs the bovine prion MADCOW has ten sequential GxxxG sequences, while Alzheimer's beta-amyloid contains four.[294,295]

Comirnaty and Spikevax used a **modified RNA nucleoside** N1-methyl pseudouridine (Ψ) to replace uracil (U→Ψ),[296] which according to FDA briefing documents was done to reduce activation of the innate immune system and augment spike protein expression (pg.16).[297] Their prion potential arises because RNA molecules containing this modified RNA nucleoside can cause **altered secondary structures** and face **codon reading-stopping issues**.[298] The literature indicates COVID-19 vaccine mRNA contains sixteen UG tandem repeats (ΨGΨG), additional UG (ΨG) rich sequences, and two GGΨA nucleotide sequences (G: guanine, U: uracil, A: adenosine). As such, mRNA vaccines could theoretically induce RNA-binding proteins like TDP-43 and Fused in Sarcoma (FUS) to fold into their pathologic prion conformations.[299,300]

The exosome shedding potential of SARS-CoV-2 spike proteins raises two prion-related issues. Firstly, prion proteins can self-replicate by acting as templates for converting other copies of the same protein.[301] Secondly, the transmission of recombinant prion proteins or infected tissue from prion diseased animals leads to prion disease in transfected animals.[302,303,304,305,306] This highlights a seemingly unassessed potential for transmissible prion diseases via spike protein exosome shedding. Exosomes have been confirmed in **all bodily fluids** such as epithelial secretions, saliva, urine, mucous, respiratory secretions during respiratory disease, blood, breast milk, cerebral spinal fluid, and amniotic fluid.[307,308,309,310,311] Pfizer understood the potential shedding risk and harm because it anticipated the possibility of secondary exposure to Comirnaty associated with pregnancy, breastfeeding, and in the workplace via inhalation or skin contact (Study protocol section 8.3.5).[312] No shedding results were disclosed in the prior cited regulatory review documents supporting Comirnaty's EUA approval. Thus, exosome shedding of COVID-19 spike proteins around booster times could represent a potential **unassessed environmental hazard** in the workplace, school, home, and the general environment.[313,314,315]

1.4 Regulatory Reviews Indicate Critical Deficits in our Preclinical Safety Understanding for Comirnaty at EUA Approval (USA, EU, & Australia)

The following cited FDA (USA),[316] European Medicines Agency (EMA, EU),[317] and Therapeutic Goods Administration (TGA, Australia)[318] regulatory documents supporting Comirnaty's EUA approval were pooled and reviewed, among others specifically cited. This was done to identify what was missing from Comirnaty's preclinical safety assessment that if it were present would have enabled a broader understanding of vaccine safety before mass vaccination using a first-in-class novel gene-therapy-vaccine technology. Red flags were raised in my review of these overseas regulatory assessments for Comirnaty, which had numerous predictable safety risks (i.e., *ADE, antigenic imprinting, virus-free spike protein-*

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related pathologies, lipid nanoparticle chemical toxicity, genotoxicity, and fertility-reproductive issues). The glaring deficits (to me) left me with **two rhetorical questions**: (1) Was a broader repertoire of preclinical safety studies conducted but not provided by the regulators within their regulatory reviews? (2) Did the regulators provide a low hurdle for preclinical safety assessment for Comirnaty?

1.4.1 Biodistribution Studies Bypassed Spike Protein-ACE2 Pathology Mechanisms

Critically, for biodistribution studies, the spike protein-encoding mRNA-LNP was **substituted** with a surrogate luciferase expressing mRNA-LNP using various routes of administration. Consequently, the regulatory reviews did not disclose Comirnaty-specific data about the tissue distribution and kinetics of spike protein mRNA and its transcribed spike protein after vaccination. As such, regulators seemingly had a **no-to-little understanding** of Comirnaty's spike protein pharmacokinetics and any pathologies triggered by its **high-affinity binding** with ACE2 receptors distant from the injection site in a relevant species.

In my opinion, using rats in the toxicology studies would likely have flattered Comirnaty safety's profile. This reflects the **low-affinity binding of rat ACE2 receptors** to the spike protein in ACE2-rich tissues like cardio-/cerebro-/respiratory-/renal- vascular endothelium, cardiac muscle, alveoli, brain, gastrointestinal, gonads, etc. After all, we knew that the SARS-CoV-1 (2002) spike protein receptor binding domain bound the mouse ACE2 receptor with lower affinity than human ACE2 and that binding with rat ACE2 was even lower affinity (i.e., *at near background levels*).[319,320] A more relevant species for toxicology assessment would have been human-ACE2 transgenic mice,[321,322] and non-human primates due to their higher human-ACE2 homology and greater binding affinity with SARS-CoV-2.[323,324]

Crucially, the **intravenous (IV) route** of administration for Comirnaty was not detailed in the regulatory review documents. Instead, Comirnaty's LNP-spike protein mRNA was **substituted** with a single intravenous dose (IV) study in rats using an LNP-luciferase-mRNA (i.e., *avoiding ACE2 interactions mediated by furin*). The IV route of administration of the spike protein encoded mRNA to mice could have been used to mimic accidental IV administration in humans, which might have identified **acute myopericarditis** as a potential Comirnaty risk and directed the use of cardiac, endothelial, and blood clotting biomarkers for the clinical studies.[325]

1.4.2 Pre-Clinical Toxicology Revealed a Debilitating Proinflammatory Response, BUT the Lipid Nanoparticle Formulation Without mRNA went Unassessed

From a safety perspective, modified mRNA delivered in lipid nanoparticles (LNP) is a complex molecule that requires the safety assessment of Comirnaty, LNP delivery system, LNP components, and manufactured spike proteins to fully understand the pharmacokinetics (i.e., *absorption, distribution,*

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metabolism, excretion), pharmacodynamics (i.e., *biochemical and physiologic effects*), and the safety and toxicology.[326] The regulatory reviews for Comirnaty's pharmacokinetics **did not fully detail** Comirnaty or its LNP formulation or LNP components (i.e., ALC-0159 and ALC-0315). Thus, regulators could not determine what happened to the spike protein mRNA, LNP chemicals, or the manufactured spike protein after vaccination. There was also no repeat dose toxicity assessment provided in the regulatory review documents for Comirnaty's LNP formulation or its novel ingredients, making it impossible to discern if the observed pro-inflammatory response was due to the LNP formulation or a result of mRNA immunogenicity.

Only one species (i.e., healthy young rats) was used in repeat dose toxicity testing using Comirnaty and other COVID-19 mRNA-LNP variants, which according to the TGA, was adequately justified by Pfizer. This regulatory acceptance came despite the well-known issues linked to rat ACE2's low-affinity binding of the humanized spike protein and the pro-inflammatory nature of LNP formulations. This rat toxicology study involved three intramuscular doses one week apart, with a three-week recovery phase, and was conducted without any dose escalation. The TGA considered the one-week interval **non-optimal** because the immune response takes 2-3 weeks to reach its peak. Comirnaty's novel lipid excipients had **long liver retention times** (EMA: ALC-0315 6-weeks, ALC-0159 > 2 weeks) and three weeks was the planned clinical study booster interval. Nonetheless, the TGA, and other regulators, permitted this.

The overwhelming findings of the toxicology studies were that of a robust **pro-inflammatory response,** which led to fever and a statistically significant reduction in rat body weights nine days after vaccination, indicating **significant systemic illness**. The associated pathologies included injection site inflammation and abnormal clinical pathologies (i.e., *moderate-strong leukocytosis, strong transient lowered reticulocytes and moderate reductions in red blood cell parameters, raised fibrinogen levels, significant increases in acute phase proteins, a decreased albumin/globulin ratio, and significant liver enzyme elevations*) and histopathologies (i.e., *hyper-cellularity of draining lymph nodes, spleen and bone marrow, and reversible portal hepatocyte vacuolation probably linked to the LNP lipids*). The above changes were partially or fully reversed within the 3-week recovery phase.[327] These results were consistent with other preclinical studies, which described LNPs used to deliver mRNA as highly inflammatory.[328]

The TGA commented that treatment-related microscopic findings were seen at the injection sites and in surrounding tissues, draining lymph nodes, bone marrow, spleen, and liver, which were "*consistent with immune responses and inflammatory reactions*." Given the widespread distribution of radiolabeled LNP mRNA luciferase at 48 hours post-vaccination in the rat body, it was surprising that histopathology, immuno-toxicology, and bio-marker information was **not provided** in any of the regulatory reviews for blood vessels, heart, brain, lungs, kidney, intestinal tract, endocrine glands, gonads, and placentas.

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1.4.3 Pre-Clinical Assessment of Autoimmunity, Genotoxicity, and Carcinogenicity was Not Detailed in the Regulatory Reviews

Surprisingly, there was no study information provided in the three regulatory reviews regarding autoimmunity, genotoxicity (i.e., an ability to cause genetic alterations), and carcinogenicity for Comirnaty as a first-in-class mRNA gene-therapy-vaccine. The absence of autoimmune disease information raised a red flag for me because this fundamental lead optimization research science should have been conducted to predict human vaccine safety, develop clinical assays, and support regulatory submissions and investigator brochures. Furthermore, the failures of SARS-CoV-1 and MERS coronavirus spike protein vaccine prototypes putatively involved a pathogenesis mechanism consistent with autoimmunity in lung tissues.[329]

The SARS-CoV-2 spike protein encoded by Comirnaty's mRNA could have been computationally checked for its sequence homology and cross-reactivity potential with human tissue proteins, among other related predictive assessments. Using in-vitro assays with an array of SARS-CoV-2 spike protein monoclonal antibodies and tissue antigens would then have been the next logical step for assessing autoimmunity potential. This research was conducted by independent researchers who **demonstrated a high degree of cross-reactivity** between SARS-CoV-2 spike protein and tissue antigens and, therefore, the potential for autoimmunity with COVID-19 infection (section 1.3.3) and, by implication, with vaccination.

Of great genotoxicity concern is that it has recently been discovered that SARS-CoV-2 uniquely possesses a functional **nuclear localization signal** (NLS) motif "PRRARSV" between the spike protein S1 and S2 sub-units. This functional NLS motif enables SARS-CoV-2 spike proteins to **translocate into the nucleus** in infected airway epithelial cells and to potentially shuttle spike protein **mRNA,** and possibly the whole genome, into the nucleus (preprint).[330] Its role in pathogenesis is not publicly understood yet. This NLS motif also comprises the uniquely encoded Arginine-doublet containing furin cleavage site (FCS, "PRRA") used by SARS-CoV-2 to gain ACE2-mediated cell entry. Furthermore, a 19-nucleotide genome portion comprising this 12-nucleotide FCS was 100% matched with a patented **reverse complement artificial sequence**. This Moderna patent covers oncology-related polypeptides containing an FCS and microRNA seed-complementary sites comprising 19-25 nucleotide non-coding RNAs containing a seed region, which is the complement to a target sequence that can be used to **down-regulate gene expression** (cited in section 2.2.1). Adding further concern to genotoxicity potential are two recent controversial publications that confirm Comirnaty's modified mRNA and SARS-CoV-2's RNA are **reverse-transcribed into DNA** in human cells in-vitro.[331,332]

As a **general comment**, while there may be no-, little-, or controversial- literature to support or refute spike protein mRNA reverse transcription and even its genome integration and gene transfer potential,[333,334] or the confirmed biological role of nuclear localization signal and microRNA seed-complementary sites, this

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may reflect that research was not permitted to be conducted or be published. This, in my view, would be typical of industry and science paradigms being centrally controlled (i.e., COVID-19, climate change, geoengineering climate change, fossil fuel reserves). That is not evidence **that such things don't occur**.

1.5 The Clinical Efficacy and Safety Claims for Comirnaty at EUA Approval Were Falsifiable

Reviewing the overseas population-level vaccine effectiveness and safety-mortality data in 2022 behooves us to explain the **vast chasm** between what has arisen in 2022 versus the claimed 95% efficacy and safety narrative touted with the first Emergency Use Authorization (EUA) of COVID-19 vaccines. In my opinion, on the 2020 side of this chasm of difference for Comirnaty (i.e., *my primary focus for reasons detailed in the Section 1 introduction*) lays a claim of 95% vaccine efficacy and vaccine safety that was **falsifiable-refutable** from the outset, based on: **(1)** the use of high false-positive diagnostic methods to diagnose clinical study cases without whole viral genome sequencing or viral culture to confirm the presence of a live-whole virus, **(2)** investigator discretion to exclude significant amounts of uncertain or non-specific efficacy data from the efficacy calculation without apparent remediation or explanation, **(3)** a clinical study that did not provide evidence of biomarker monitoring to assess predictable pathology mechanisms, **(4)** clinical study inclusion criteria that did not prioritize the most appropriate at-risk population suffering comorbidities involving ACE2/furin rich tissues-organs associated with severe COVID-19 outcomes.

The **FDA, and other drug regulators, held overall responsibility** for accepting the Phase 3 study design and confirming that Comirnaty was safe and efficacious before EUA approval. I had **ZERO confidence** in the assured 95% vaccine efficacy and safety conclusion.

1.5.1 How Comirnaty's Falsifiable 95% Vaccine Efficacy Was Determined

Based on Comirnaty's interim safety (i.e., a median of two months) and efficacy data underpinning its EUA approval, Pfizer published data indicated Comirnaty was safe and 95% efficacious at preventing laboratory-diagnosed symptomatic COVID-19 disease from 7 days after dose 2 to the end of the surveillance period (i.e., a mean of 46 days surveillance). This claimed 95% relative vaccine efficacy (i.e., *RVE = 1 - vaccinated risk / unvaccinated risk, vaccinated risk = 8 cases / 18,198 vaccinated, unvaccinated risk = 162 cases / 18,325 placebo group*) corresponded with an absolute risk reduction of 0.84% (i.e., *ARR% = unvaccinated risk - vaccinated risk*) or 840 fewer cases per 100,000 population. This data and calculations were derived from Table 2 in Pfizer's published results.[335]

I believe the claimed 95% vaccine efficacy always needed to be scrutinized and treated **with caution**. This caution was merited because the mean surveillance period was only 46 days (i.e., during peak immunity),

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and the risk of predictable negative vaccine efficacy via antibody-dependent enhancement of viral infection and vaccine failure via antigenic imprinting was always going to increase as **immunity waned** and with the later emergence of **antigenically distinct strains** like Delta and Omicron. This caution on vaccine efficacy also applied an understanding of **coronavirus (i.e., ADE/VAED**) and general vaccine biology (i.e., **antigenic imprinting**), and **viral mutation** (i.e., associated with pandemic waves), or knowledge that has been in existence for decades in industry and academia. This **would/should** have been known by anyone claiming or relying on coronavirus vaccine expertise (i.e., *R&D program leaders, regulatory specialists, national and international vaccine advisory board experts, government vaccine advisors, etc*.).

Peter Doshi, assistant professor of pharmaceutical health services at the University of Maryland and senior editor for the British Medical Journal, critiqued the 95% Comirnaty efficacy calculation.[336] Accordingly, there were 3,410 "suspected but unconfirmed COVID-19 cases" (i.e., "*symptomatic COVID-19 that were not PCR confirmed*") that were not included in the efficacy calculation. In justifying the 3,410 suspected but unconfirmed COVID-19 cases, the FDA states, "*It is possible that the imbalance in suspected COVID-19 cases occurring in the 7 days post-vaccination represents vaccine reactogenicity with symptoms that overlap with those of COVID-19*" (pg.42, FDA review).[337] In my opinion, that *excusatory* FDA rationale did not reflect the fact that there were 1,594 suspected but unconfirmed COVID-19 cases in the vaccinated group and 1,816 in the *placebo group*.

Why were 170 cases confirmed by PCR (i.e., 8 Comirnaty + 162 placebo, or 4.7%), and yet 3,410 suspected COVID-19 cases were not confirmed by PCR or an alternative diagnostic method (i.e., 95.3%)? Why did study investigators have the discretion **not to follow up** on this high volume of cases and re-sample or use a backup diagnostic method? Why did the FDA appear to excuse this issue? "*Overall though, these data do not raise a concern that protocol-specified reporting of suspected, but unconfirmed COVID-19 cases could have masked clinically significant adverse events that would not have otherwise been detected*." According to my calculation, when the 3,410 subjects were added back, the relative vaccine efficacy was 19%, which fell **short of the 50% minimum** vaccine efficacy required for EUA.[338]

Professor Doshi raised another important efficacy issue. There was a 5-Comirnaty-to-1-placebo imbalance among 371 subjects excluded from the efficacy calculation for "*important protocol deviations on or prior to 7 days after Dose 2*" without explanation (Table 2, pg.18, FDA review). If 4/6ths of the protocol deviants (n=247) were added back to the Comirnaty data as positive cases, and Comirnaty and placebo group protocol deviants were balanced 1-to-1 (n=62 subjects each), the relative vaccine efficacy would have been **-59%** or replicating the negative vaccine effectiveness observed in 2021/2022. These issues raised Professor Doshi's concerns about the "*trustworthiness and meaningfulness of the reported efficacy results*." In my view, these issues should have been sufficient to have **prevented Comirnaty's EUA approval**.

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1.5.2 High False Positive Diagnostic Methods Generate Bogus Data (Rule of Thumb)

Comirnaty vaccine efficacy was based on real-time reverse transcription polymerase chain reaction (RT-PCR, shortened to PCR) confirmed cases, plus one or more non-specific flu- or gastrointestinal-like symptoms (i.e., applicable to COVID-19 and any other respiratory and gastrointestinal viruses, non-infectious ailments, and vaccine reactogenicity).[339] Dr. Michael Yeadon (i.e., *former Vice President & Chief Scientific Officer of Allergy & Respiratory at Pfizer Global R&D*) plus another petitioned the EMA on Comirnaty's efficacy assessment shortcomings (01/12/2020).[340] Accordingly, "*The current study designs for the Phase II/III trials of BNT162b ("the Pfizer Vaccine") are inadequate to assess efficacy accurately*."

In their EMA petition, Yeadon et al. stated, "*ACTION REQUESTED 2. Stay the Phase III trial of BNT162 (NCT04368728) until its study design is amended to provide that: Before a EUA or unrestricted license is issued for the Pfizer vaccine, or for other vaccines for which PCR results are the primary evidence of infection, all endpoints or COVID-19 cases used to determine vaccine efficacy in the Phase 3 or 2/3 trials should have their infection status confirmed by Sanger sequencing ("to confirm that the tested samples, in fact, contain a unique SARS-CoV-2 genomic RNA."), given the high cycle thresholds used in some trials."*

The PCR method used worldwide to diagnose SARS-CoV-2 from January 2020 was based on the *hastily* "peer-reviewed" Corman-Drosten protocol (i.e., 24-hours, "*Received 2020 Jan 21; Accepted 2020 Jan 22.*"),[341] which was downloadable from the WHO website before its peer review (2020 Jan 17).[342] This protocol was described as "*severely flawed*" by an international consortium of expert scientists who petitioned for its journal retraction,[343] which was accompanied by their review report, "*External peer review of the RT-PCR test to detect SARS-CoV-2 reveals **10 major scientific flaws** at the molecular and methodological level: consequences for false positive results.*"[344] According to these experts, this test method led to the "**worldwide misdiagnosis of infections attributed to SARS-CoV-2 and associated with the disease COVID-19**".

This WHO-promoted Corman-Drosten PCR reference protocol promoted a cycle threshold of 45.[345,346] According to this international consortium of experts, PCR data evaluated as positive after a cycle threshold (Ct) value of 35 cycles are *completely unreliable ("as is the case in most laboratories in Europe & the US")*,[347] because this was known to generate >97% false positive cases.[348] According to this last cited publication, "*It can be observed that at Ct = 25, up to 70% of patients remain positive in culture and that at Ct = 30, this value drops to 20%. At Ct = 35, the value we used to report a positive result for PCR, <3% of cultures are positive.*" Furthermore, a systematic review concluded that those with a high cycle threshold were unlikely to have infectious potential while reminding us that a complete live virus was necessary for virus transmission, not RNA fragments identified by PCR.[349]

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I vividly remember watching the WHO Director-General Tedros Adhanom Ghebreyesus (16/03/2020) tell the world, "*We have a simple message to all countries - test, test, test*" (i.e., using these high false positive PCR case generators).[350] Less than one year later, the WHO amended its PCR protocol recommendations to minimize the generation of false positive data (13/01/2021).[351] Unfortunately, by then, the **damage had been done** in my view because this original WHO-promoted Corman-Drosten high false positive PCR protocol had been used to diagnose the global and national cases in 2020 that were used to justify **policies**, including travel restrictions, lockdowns, social distancing, mask-wearing, workforce confinement, closure of economic activity, and subsequently in 2020-21 to induce and mandate vaccination in low-risk demographics worldwide.

In my view, this high false-positive PCR issue, which I term a **bogus COVID-19 case generator**, and failure to confirm a live virus potentially **invalidated** the Comirnaty Phase 3 clinical study efficacy data in the drastically filtered 170 subjects, without even considering the 3,410 suspected but unconfirmed cases and the biased 371 protocol deviants excluded from the efficacy calculation. In other words, I had **ZERO confidence** in Comirnaty's claimed 95% vaccine efficacy at EUA approval.

As an aside, on the 12th of February 2021, and perhaps on the 17th of August 2021, although it is unclear from the Ministry of Health's reply to freedom of information (FOI) requests,[352] **New Zealand** was still using a cycle threshold of 40 (FOI disclosures).[353] It would be interesting to know what cycle threshold was used to diagnose that one Delta case that led to Auckland's August 2021 lockdown and if an alternative diagnostic method was used to confirm the presence of a whole-live virus (and during 2022). After all, more than five million COVID-19 vaccine doses were administered during this lockdown period.[354]

1.5.3 Statistical Analysis Confirms Comirnaty was Unsafe at EUA Approval

Independent expert safety analysis of the Pfizer Phase 3 clinical safety data demonstrated that Comirnaty was unsafe at the FDA's Emergency Use Authorization.[355,356] The safety narrative that hit the world's media in December 2020 for Comirnaty was **falsifiable** and unsupported by statistical analysis.

An independent secondary analysis of serious adverse events (SAE) and Brighton Collaboration adverse events of special interest (AESI) was conducted with Comirnaty's Phase 3 clinical study data used to support its Emergency Use Authorization (EUA,[357] NCT04368728).[358] This study identified a 57% higher risk of serious AESI with Comirnaty (risk ratio 1.57, 95 % CI 0.98 to 2.54), corresponding with 10.1 additional AESI per 10,000 vaccinated (95 % CI −0.4 to 20.6). There was a 36 % higher risk of SAE (risk ratio 1.36, 95 % CI 1.02 to 1.83), which corresponded with 18.0 additional SAEs per 10,000 vaccinated (95 % CI 1.2 to 34.9). A harm-to-benefit analysis demonstrated that the excess risk of serious AESIs (10.1 per 10,000 vaccinated) was higher than the risk reduction for COVID-19 hospitalization relative to the

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placebo group (2.3 per 10,000 participants). These results raised concerns with the study's vaccine safety experts that Comirnaty was associated with more harm than the FDA had concluded at EUA (i.e., SAEs were "balanced between treatment groups").[359] In my view, their analysis using the FDA's data tables indicates the FDA **failed** to analyze the safety data appropriately, or they **inappropriately** approved it.

The largest excess risk occurred with **coagulation disorders**, while there were more **cardiovascular** AESIs in the vaccinated versus the placebo group. Further supporting Comirnaty's inappropriate EUA, the FDA detected four potential Comirnaty AESI in the elderly (≥65 years) based on medical claims data (July 2021). These AESI were *pulmonary embolism, acute myocardial infarction, disseminated intravascular coagulation* (i.e., all **coagulation disorders**), and immune thrombocytopenia.[360] Disproportionality studies comparing influenza vaccines with mRNA COVID-19 vaccines and utilizing three vaccine pharmacovigilance databases (VAERS, EudraVigilance, and VigiBase) also reported excess risks for many of the same SAEs (i.e., *cardiovascular events, coagulation events, hemorrhages, gastrointestinal events, and thromboses*).[361,362,363] Cynically, had the FDA adequately analyzed the safety data, had Pfizer prioritized the elderly in the Phase 3 study (i.e., the elderly comprised only 4% of the study – section 1.5.4), among other issues, there is a good chance Comirnaty would not have been approvable.

My analysis also utilized the interim 2-month safety data from the unblinded study period that Pfizer had used to support the EUA approval (Supplementary Tables S3 and S4[364]) and replicated another analysis.[365] Data for **related adverse events** (AEs, investigator-assessed, placebo = 1,311, Comirnaty = 5,241, 4.0x), any **severe AEs** (interfered with bodily functions, placebo = 150, Comirnaty = 262, 1.8x), any serious AEs (attended A&E or was hospitalized, placebo = 116, Comirnaty =127, 1.1x), and deaths (placebo = 14, Comirnaty = 20, 1.4x) was assessed. A Chi-square test of independence demonstrated that the observed proportion (i.e., cases divided by cohort totals, placebo = 21921, Comirnaty = 21926) of any related, severe, serious AEs and deaths was higher in the Comirnaty group than expected and lower than expected in the placebo group. These differences were **highly significant for severe and related AEs** ($p < 0.00001$).

1.5.4 Safety Consequences of the FDA-Approved Phase-3 Clinical Study Design for Comirnaty

One of the safety consequences of the FDA-approved phase-3 clinical study design for Comirnaty used to support EUA approval was that it did not provide evidence that **biomarker assays** had been used to detect **predictable pathogenesis mechanisms**. Such use could have potentially detected predictable safety issues and sub-clinical and comorbid diseases in a controlled clinical setting. According to experts, biomarker assays should have been used to detect coagulation/clotting issues (i.e., D-dimers, other), endothelial damage (i.e., occludin and claudin), inflammatory reactions (C-reactive protein, pro-inflammatory cytokines), cardiac damage (troponins), autoimmune disease markers (i.e., HMGB1, CXCL13, Dickkopf-

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1), Alzheimer markers (amyloid-beta and phosphorylated tau), etc.[366] In other words, biomarkers should have been used for predictable pathogenesis mechanisms as detailed in section 1.3. Assays should also have been used to detect ADE during the full-length unblinded clinical study period. Furthermore, in my and others' views,[367] information about the **substantial risk of ADE, VAED, and antigenic imprinting** should have been a key part of the clinical study **informed consent**.

The Canadian Covid Care Alliance (CCCA) excellently summarized what they considered were *major shortcomings* of Comirnaty's clinical studies, including those used to support COVID-19 vaccine use in young children (Pfizer: **"more harm than good"**).[368] The CCCA provided a useful alternative clinical study design, to which I have added clinical end-point suggestions, that in my opinion would have answered important outstanding efficacy and safety questions: (1) no prior exposure to COVID-19 plus Comirnaty, and (2) no prior exposure to COVID-19 plus placebo (i.e., comparative safety and efficacy assessment), (3) previously infected plus Comirnaty (i.e., to understand any efficacy benefit or vaccine-induced pathology), (4) previously infected plus placebo (i.e., comparative natural immunity protection assessment). Pfizer's omission of the last two study arms means the assessment of vaccine-induced pathologies associated with (re)infection (i.e., ADE/VAED) and a Comirnaty comparison with **natural immunity** was avoided.

In addition to assessing community-relevant disease protection endpoints (see next), the study, in my view, should have confirmed viral infection (i.e., whole viral genome sequencing, virus culture), viral loads, and viral shedding (M. Yeadon also).[369] This would have confirmed the vaccine's ability to protect against SARS-CoV-2 infection, reduce viral loads and transmission, and the duration of protection.

Based on the demographic burden of community COVID-19 disease it was the CCCA and my view that the Pfizer study did not prioritize the most appropriate **at-risk population** (i.e., the elderly with multiple comorbidities). Before the study, it was known that 95% of people who died from COVID-19 had one or more co-morbidities, with an average of four.[370] We also knew 85% of the people most at risk from COVID-19 were over 75yrs old (sections 1.1.2-5).[371] Sections 1.1.3-4 confirm the elderly risk factor, as the vaccinated elderly accounted for most COVID-19 deaths and hospitalizations. Section 1.3.3 Mechanism 1 confirms the most prevalent **comorbidities** and biggest risk factors associated with severe COVID-19 outcomes (i.e., elderly, comorbidities associated with the cardiovascular, respiratory, brain, and kidney systems, obesity, and diabetes). However, **the elderly were not prioritized** in Pfizer's Phase 3 study because those aged older than 75 years represented only 4% of study subjects,[372] while only 21% of subjects had one or more comorbidities. Instead, younger demographics were prioritized that would be less likely to need Comirnaty or suffer an adverse event (i.e., have fewer preexisting or comorbid diseases).[373]

An important consequence of **unblinding** the Phase 3 clinical study for Comirnaty **28 months early** (i.e., Comirnaty was given to the placebo group) after EUA was it **eliminated** the possibility of detecting

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potential **statistical differences** in vaccine efficacy and vaccine-induced pathologies and ADE between the Comirnaty and the placebo groups. In my view, this will undermine Comirnaty's full Phase 3 safety assessment, which made me highly suspicious when this news was announced. The primary safety end-points for Comirnaty in my view should have been both **infection and transmission prevention** and all-cause **disease morbidity** pooling both COVID-19 disease, death, and vaccine adverse events from day 0 (i.e., *A&E visit, hospitalization, duration of illness, symptom scores*, etc.) in a study that prioritized the elderly with comorbid disease associated with severe COVID-19 outcomes (i.e., those most at risk, section 1.3.3 Mechanism 1).

As a consequence, had the FDA insisted that Pfizer; prioritize the elderly with multiple most-prevalent comorbidities associated with severe COVID-19 outcomes and use biomarkers in its Phase 3 Comirnaty study, and had it not permitted Pfizer to unblind the study 28 months early, then the **ability to deny** vaccine-induced harm or record vaccine-associated deaths as **not attributable** to vaccination post-EUA could have been prevented (i.e., by **healthcare agencies, coroners, and pharmacovigilance units**). However, it was the FDA and other regulators who permitted these Phase 3 safety monitoring shortcomings.

2 SARS-CoV-2 FEATURES INFECTIVITY-ENHANCING GAIN-OF-FUNCTION TECHNOLOGY UNPRECEDENTED IN NATURE

There are two main reasons for detailing a putative coronavirus gain-of-function origin to SARS-CoV-2 and highlighting other potential origins beyond the Wuhan Institute of Virology (Wuhan-IV). Firstly, the high infectivity in the unvaccinated before EUA of any vaccine (12/2021) and the **enhanced rates of infection** in the vaccinated putatively via antibody-dependent enhancement of virus infection could have been facilitated by a **genetically modified** SARS-CoV-2 spike protein (i.e., **receptor binding domain, and N-terminal domain**). Secondly, the world is **left vulnerable** to future pandemics if there was no accidental release of a SARS-CoV-2 precursor from the Wuhan-IV.

This section details the lack of hard evidence for a zoonosis, the unprecedented gain-of-function features without evolutionary precedent in SARS-CoV-2, the SARS-CoV-2 origin cover-ups, and the main gain-of-function research networks and funders involved. This gives insight as to other potential origins for SARS-CoV-2 beyond the Wuhan-IV. Other origin possibilities exist beyond those reviewed, meaning that what follows is not definitive or accusatory and is provided on an information-sharing basis. In my view, this is uncommon knowledge that the mainstream media and government narrative have bypassed. Thus, if a Pentagon- and NIH-funded coronavirus gain-of-function and zoonosis expert was **exposed for trying to cover up** SARS-CoV-2's gain-of-function origin, then he and his connections one degree removed were

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justifiably scrutinized below (i.e., *Wuhan-IV, the University of North Carolina at Chapel Hill, Metabiota, and the WHO*) to see what could be learned. I concluded that the origin of COVID-19 was not investigated as though it was a **potential international crime scene**.

Two US House Foreign Affairs Committee Report Minority Staff investigation reports (HFACR), "*The origins of the COVID-19 global pandemic, including alleged roles of the Chinese Communist Party and the World Health Organization*,"[374] and "The origins of *COVID-19*: an investigation of the Wuhan Institute of Virology,[375] as well as COVID-19 origin evidentiary publications are very *informative references*.[376] However, in my view, while the HFACR reports provide many factual data points, information, and evidence, the conclusions may have been politically motivated and do not reflect the **confounding issues**.

2.1 There Is Zero Hard Evidence for a SARS-CoV-2 Zoonosis (Virus Progenitor or Animal Host)

A significant evolutionary gap exists between SARS-CoV-2's closest known coronavirus 2B-lineage bat origin RaTG13 and BANAL-52 strains (i.e., ~ 96% average genetic similarity),[377] which represents decades of evolutionary divergence.[378,379] A coronavirus isolated from Malayan pangolins shared the same spike protein receptor binding domain (RBD) sequence as SARS-CoV-2, indicating pangolins could potentially have acted as an intermediate SARS-CoV-2 host between bats and humans until more closely scrutinized. Even though pangolin coronaviruses have a high spike protein RBD sequence similarity to SARS-CoV-2, their whole genome is only ~ 90% similar.[380] However, unlike SARS-CoV-2, all pangolin-CoVs identified to date lack a uniquely encoded Arginine-doublet containing furin cleavage site between S1 and S2 sub-units. At the same time, no progenitor virus has been found in pangolins. This suggests pangolins were not the origin of SARS-CoV-2.[381]

Despite extensive sampling of animals at the Huanan seafood market and with its market suppliers and the testing of **80,000 wildlife (>200 animal species**, including pangolins), livestock, and poultry samples from 31 provinces in China, none tested positive for the virus and/or SARS-CoV-2-specific antibodies.[382] In my view, in the absence of a genetically closer zoonotic SARS-CoV-2 progenitor and associated animal host, there is **zero evidence for a zoonosis** cause of the COVID-19 pandemic.

According to the scientist famed for identifying SARS-CoV-2, Dr. Zheng-Li Shi, its closest relative was RaTG13, a bat coronavirus, which was 96.2% identical at the whole genome level with SARS-CoV-2. The spike protein encoded S gene for SARS-CoV-2, and RaTG13 are longer than other bats' SARSr-CoVs, indicating a potential link. However, RaTG13 does not possess a furin cleavage site, a SARS-CoV-2-like receptor binding domain (RBD), and it does not bind to human ACE2 with high affinity.[383] The major differences in the S gene between SARS-CoV-2 and SARS-CoV-1 were three short insertions in the N-

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terminal domain and changes in four out of five key residues in the receptor-binding domain.[384]

Dr. Shi's time-intensive research findings claiming a bat zoonosis origin were submitted to the Nature journal the same day China's National Health Commission confirmed human-to-human transmission (20/01/2020). Dr. Shi was forced to publish an addendum to their bat-zoonosis origin **narrative-leading publication** revealing that RaTG13 was ID4991,[385] which Shi et al. had discovered in 2012-2013.[386] In reality, the full genome sequence was obtained in 2018 and not as stated in January 2020. With that misstep, was Dr. Shi covering something up, and was this relevant to SARS-CoV-2 origin? The following preprint details the contentious issues necessitating Dr. Shi's addendum and concluded, "*This paper was rushed to make a premature connection between bat coronavirus and SARS-CoV-2, drawing a potential bat origin scenario to support SARS-CoV-2 zoonotic transmission from bat to human.*"[387]

In the absence of a genetically similar bat SARSr-CoV (i.e., >>96.2%) and a confirmed bat host harboring that SARS-CoV-2 progenitor virus, it is important to understand how Dr. Shi et al. created that **speculative zoonosis link** to bats and the local Huanan seafood market. Firstly Dr. Shi stated, "*Previous studies have shown that some bat SARSr-CoVs have the potential to infect humans*." Two of three studies cited in support of that quoted statement were based on coronavirus gain-of-function research using human cell lines expressing ACE2 receptors conducted by Wuhan-IV, the University of North Carolina at Chapel Hill, and others, with Ralph Baric as the corresponding author in both cases.[388,389] The third cited publication created the link between bat SARSr-CoVs and a SARS-CoV-1 zoonosis (ACE2 receptor-mediated), which was affiliated with Wuhan-IV and with Dr. Shi as the corresponding author and Dr. Daszak, among others.[390] The second speculative zoonosis link was created by the following uncertain-vague quote, "*It appears that most of the early cases had contact history with the original seafood market.*"

In my view, these prior cited publications **provide zero hard evidence** of a Huanan market bat zoonosis, but rather highlight the **key protagonists** in a US-China coronavirus gain-of-function research network. The Wuhan Institute of Virology (Dr. Shi et al.[391]) and other Research Institutes in China, and the University of North Carolina at Chapel Hill (UNCCH, Dr. Ralph Baric et al.[392,393,394,395]) represented known global epicenters for coronavirus gain-of-function research, in which Dr. Daszak collaborated (publication list[396]).[397,398,399,400,401,402,403] The NIH has funded Dr. Baric's gain-of-function, zoonosis, and his other research to the tune of $160-plus million since 1986, making him a coronavirus gain-of-function expert with few peer.[404] Much of this collaborative network's research was focused on modifying the spike protein of coronaviruses that **could not infect humans so that they could** without the need for a zoonotic event.

2.2 SARS-CoV-2 Spike Protein Bristles with Gain-of-Function Technology that Enabled-Enhanced Human Infectivity and Pathogenicity

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Researchers examined how SARS-CoV-2 spike protein binds to the ACE2 receptor of various animal species to understand the SARS-CoV-2 potential species of origin. Surprisingly, they found that SARS-CoV-2's spike protein binds the strongest to human ACE2 receptors (December 2019 strains, human> pangolin> dog> monkey> hamster> ferret> cat> tiger> bat> civet> horse> cow> snake> mouse). Typically, a zoonotic virus exhibits the **highest binding affinity** initially for its **originating host species** and lower initial affinity for receptors of the new host species until it mutationally adapts. This study's results suggest that SARS-CoV-2 spike RBD evolved by selection on a human-like ACE2, not a pangolin, bat, or mouse ACE2 receptor.[405] The first preprint version of this paper went further, concluding, "*the data indicate that SARS-CoV-2 is uniquely adapted to infect humans, raising important questions as to whether it arose in nature by a rare chance event or whether its origins might lie elsewhere.*"[406] A potential genetic-engineering origin was initially proposed for SARS-CoV-2 in an FOI-disclosed email to NIH Director Dr. Anthony Fauci (pg.3187, 31/01/2020),[407] but this provisional opinion changed for their prestigious Nature Medicine publication "The proximal origin of SARS-CoV-2".[408]

Any claim that SARS-CoV-2 could not have been human-made because there were no genetic modification markers is without merit on two counts. Firstly, Dr. Ralph Baric et al. had created a method that supposedly **left no trace of genetic modification** as early as 2005, and by 2016 Wuhan-IV scientists had acquired that capability (2001-2016).[409,410,411,412,413] SARS-CoV-2's furin cleavage site and other functional sequences described below could have been added using established patented know-how (2005),[414] combined with the prior cited traceless genetic modification and recombinant coronavirus methods, and other patented methods covered by 4,000+ coronavirus patents.[415] Secondly, according to a recent preprint, the SARS-CoV-2 genome contains a pattern of **unique restriction endonuclease recognition sites**, which permit efficient dis- and re-assembly of a viral genome typical of a reverse genetic system and synthetic virus. It was concluded that SARS-CoV-2 was probably an infectious clone **made in the lab**.[416]

SARS-CoV-2 spike protein and its receptor binding domain (RBD) have some unique biomolecular features **without evolutionary precedent** among **(1)** all B-lineage beta-coronaviruses (i.e., *furin cleavage site, or FCS*), or **(2)** all coronaviruses (i.e., *HIV-1 sequences*), or **(3)** in all viruses, and all viruses that are known to contain a furin cleavage site (i.e., *A 2-in-1 FCS comprising a uniquely encoded Arginine doublet contained within a longer nuclear localization signal motif. A Moderna patented artificial sequence containing a 19-nucleotide sequence, which is the reverse complement of a virus-unprecedented 19-nucleotide sequence containing and flanking the FCS.*). These features (cited below) increased human-ACE2-receptor binding affinity 10-20-fold over the human-infecting SARS-CoV-1 (2002) and were critical to its infectivity and pathogenicity.[417,418] The spike protein and its RBD is the same part of the virus that researchers like Dr. Peter Daszak (EcoHealth Alliance), Dr. Ralph Baric and colleagues, and Dr. Zheng-Li Shi and colleagues, among other researchers, were **genetically modifying** and replacing since at least 2015.

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2.2.1 SARS-CoV-2 Spike Protein's Unique 2-in-1 Furin Cleavage Site and Nuclear Localization Signal are Unprecedented in Nature and Potentially Infringe Patents

The addition of a furin cleavage site to SARS-CoV-2's progenitor was potentially inspired by its natural use by highly pathogenic HIV, Ebola, and Marburg viruses.[419] Furin is a cell membrane-bound protease utilized by SARS-COV-2 to cleave the spike protein S1 and S2 sub-units at the FCS to activate ACE2-mediated cell entry. Furin determines SARS-CoV-2 species range, human transmissibility, [420] and pathogenesis.[421,422] **Increased serum furin levels** are evident in obese and diabetic patients, males, and **the elderly**, which are among the **most prevalent comorbidities** and biggest risk factors associated with severe COVID-19 outcomes. Thus, the spike protein pathogenesis mechanisms reviewed in section 1.3.3 Mechanism-1 place upregulated furin and ACE2-receptors plus prevalent comorbidities in tissues and organs common to all three factors **center stage. At the same time,** SARS-CoV-2 provided its genetically inserted furin cleavage site, among other features, to catalyze and enhance infectivity and pathogenicity.

In 2018 Dr. Daszak (EcoHealth Alliance) tried to obtain funding from the US Defense Advanced Research Projects Agency (DARPA) for creating genetically modified bat SARS-related coronaviruses with spillover potential (SARSr-CoV, Project DEFUSE). Dr. Daszak **sought to graft human-specific** protease cleavage sites into bat SARS-like coronavirus spike proteins and evaluate their growth potential in human cell lines after he had analyzed "*all SARS-CoV S gene sequences for appropriately conserved proteolytic cleavage sites in S2 and for the presence of* ***potential furin cleavage sites.***" Dr. Daszak planned to create and assess multiple human codon-optimized SARSr-CoVs, including **with receptor binding domain and N-terminal domain modifications**, for changes in their infectivity and pathogenicity (pg.13, 17). He planned to subcontract work to Drs. Baric and Shi, among others (pg.3).[423] Dr. Daszak's project DEFUSE funding application was rejected by DARPA due to their safety concerns because it involved gain-of-function/dual-use research; "*EcoHealth Alliance unsuccessfully proposed the use of bat SARSr-CoV backbones and not the human evolved SARS-CoV in what looks like a* **deliberate attempt** *at circumnavigating the restrictions of the P3CO framework and related DURC restrictions*".[424]

SARS-CoV-2's FCS comprises an Arginine double-codon (CGG.CGG) within a 12-nucleotide sequence encoding Proline-Arginine-Arginine-Alanine (CCT.CGG.CGG.GCA → PRRA). While an FCS is **without evolutionary precedent** in all B-lineage beta-coronaviruses,[425,426,427,428,429] the **CGG-CGG encoded** Arginine doublet containing FCS is unprecedented in **all known viral FCS**. Furthermore, out of the 42 Arginine amino acids in the SARS-CoV-2 spike protein, only two Arginines are encoded by the CGG codon, which is those encoding the FCS.[430] This probably rules out a virus recombination as the mechanism for FCS acquisition by SARS-CoV-2 and suggests either a human genome origin (i.e., mechanistically-theoretically possible) or origin by **a human specifically wanting** a CGG-CGG encoded Arginine FCS.

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The plot thickens; A 19-nucleotide genome portion that includes and flanks both sides of the 12-nucleotide FCS was 100% matched with a patented reverse complement artificial sequence **owned by Moderna** (i.e., US 2016 patent US9587003B2).[431] This patent was a continuation of four other patents dating back to 2013.[432] Common to all five patents are oncology-related polypeptides that comprise at least one protein cleavage signal and/or site, which specifies the use of a **furin cleavage site**. This patent also covers **microRNA seed-complementary sites** comprising 19-25 nucleotide-long non-coding RNAs containing a seed region, which is complementary to a target sequence that can **down-regulate gene expression**. Moderna's patented sequence listing in US9587003B2 revealed an **artificial** sequence fragment comprising 5′-CTACGTGCCCGCCGAGGAG-3′ (nt 2733-2751 of SEQ ID11652, Ambati et al.).[433,434] This is the **reverse complement** of CTCCTCGGCGGGCACGTAG,[435] which is 100% matched with SARS-CoV-2 Wuhan-Hu-1 strain from nucleotides 23601-23619 that encodes the PRRA furin cleavage site (CCT.CGG.CGG.GCA).[436] This 19-nucleotide sequence is **without precedence** in any mammalian or viral genome in the BLAST database except in SARS-CoV-2. The probability of this sequence being randomly present in a 30,000-nucleotide viral genome was estimated at 3.21×10^{-11} (Ambati et al.).

Furthermore, this "PRRA" furin cleavage site (cell entry) is subsumed within a longer functional **nuclear localization signal** (NLS) sequence, "PRRARSV." This NLS sequence enables SARS-CoV-2 spike proteins to **translocate into the nucleus** in infected airway epithelial cells. It may also shuttle spike **mRNA into the nucleus** and possibly the whole genome. This nuclear transfer of spike protein is **without precedent** in all coronaviruses and represents a novel pathogenic feature (preprint).[437] Thus, an unprecedented CGG-CGG encoded Arginine doublet containing FCS subsumed within a longer NLS sequence, which is unprecedented in all known viruses, collectively provides a 2-in-1 enhanced infectivity and pathogenesis mechanism. What are the odds? Nature or Gain-Of-Function?

If future COVID-19 multi-strain vaccines still contain the Wuhan-Hu-1 strain, consider the technology motive. Vaccine design and antigen composition must always be scientifically justified. The Wuhan-Hu-1 vaccine strain's ability to protect against infection or disease is/will be marginal-negative with antigenically distinct strains (i.e., Omicron variants, new variants of concern). New vaccine strains will also likely face antigenic imprinting issues undermining any disease protection benefits (section 1.1.8). Wuhan-Hu-1's future vaccine inclusion will ensure this non-mutated multi-functional PRRARSV infectivity-pathogenicity enhancing sequence is retained, along with the other un-mutated spike protein gain-of-function infectivity-pathogenicity enhancing sequences. Be **highly suspicious** of Wuhan-Hu-1's future vaccine inclusion.

2.2.2 SARS-CoV-2 Contains HIV-1 Sequences and Utilizes the Same Lymphocyte Entry Pathway as HIV-1, Among Other Cell Entry Mechanisms Besides ACE2

Two research groups identified HIV sequences in SARS-CoV-2. One group identified four HIV-1

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insertions (i.e., gp120, Gag) in the spike protein (see PDF – obtained before its *censorship*[438]). These sequences are **without evolutionary precedent** in all coronavirus lineages. Despite these insertions being non-sequential in the primary sequence, 3D modeling revealed they helped form the receptor binding domain. The authors stated, "*It is unlikely that all 4 inserts in the 2019-nCoV spike glycoprotein fortuitously match with 2 key structural proteins of an unrelated virus (HIV-1)*.[439] Another group, including Nobel Laureate Luc Montagnier (i.e., HIV discoverer), found that 2.5% of the SARS-CoV-2 Wuhan genome comprised 16 HIV1, HIV2 and SIV fragments 18-30 nucleotides long from Env, Pol, and Integrase genes. Twelve of these fragments were concentrated in the ORF1ab and spike protein genes. The authors suggested that the genome **had been modified**.[440,441]

These HIV-1 gp120 insertions might explain SARS-CoV-2's ability to infect activated CD4 + T-lymphocytes utilizing the lymphocyte function-associated antigen-1 (LFA-1) pathway, resulting in programmed cell death or apoptosis. This LFA-1 entry pathway is independent of the conventional spike protein-ACE2 receptor cell entry pathway.[442] Interestingly, Dr. Zheng-Li Shi of the Wuhan Institute of Virology is a co-author of this prior-cited LFA-1 publication. The LFA-1 pathway is the same pathway HIV-1 uses to infect activated CD4+ T-lymphocytes, which ultimately causes acquired immune deficiency syndrome or AIDS.[443,444] Severe COVID-19 diseases were associated with a marked reduction of lymphocytes (i.e., lymphopenia) in 60-70% of patients admitted to the hospital, while fatal infections were associated with more severe and progressive lymphopenia.[445,446] If this **CD4 + T-lymphocyte entry pathway** has validity, one could *speculate* on a scenario where SARS-CoV-2-infected people could develop an AIDS-like condition in the longer term (i.e., time will tell).

2.2.3 SARS-CoV-2's Period of Intense Evolution in Mice Resulted in Omicron (Transgenic Mice?)

Alarmingly, there is also molecular evidence the progenitor of SARS-CoV-2 **Omicron** "*jumped*" from humans into mice around mid-2020, rapidly accumulating an unprecedented level of **45-point mutations** molecularly consistent with a period of intensive mouse evolution before "*jumping*" back into humans with enhanced human infectivity and transmissibility potential.[447] Were these mutations driven by **transgenic mice** expressing human ACE2 receptors, similar to those cited?[448,449] In consequence, the Omicron receptor-binding domain (RBD) now binds to the human ACE2 receptor with 2.4x the affinity of the original Wuhan-Hu-1 strain. At the same time, RBD-specific neutralizing antibodies have reduced binding affinity. There is also evidence of fundamental changes in the Omicron cell entry process.[450] In my view, if this research was valid in its conclusions, it raises big questions about someone manipulating **this pandemic**.

Dr. Daszak also collaborated with Wuhan-IV (NIH co-funded research) to develop methods for **inhibiting protective immune responses** toward bat SARSr-CoV by incorporating the immunomodulatory ORFX

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accessory protein. This increased their infectivity and pathogenesis. This genetic modification was done without leaving a molecular trace in the recombinant viral genome.[451] According to a US House Foreign Affairs Committee Minority Staff investigation, four traceless recombinant viral strains "*were tested for ACE2 utilization by these strains to infect human cell lines, civets, and bats*," citing Zeng's doctoral thesis.[452] Gain-of-function scientists also trained the SARS-CoV-2 human fitness by using human ACE2 receptor expressing in-vitro systems (i.e., *serial passage*) and transgenic mice (humanized-ACE2).[453,454,455]

2.3 Cover-Ups and Failures to Properly Investigate SARS-CoV-2's Origin

Emails obtained under FOI by US Right to Know show that a statement in The Lancet authored by 27 prominent public health scientists condemning "*conspiracy theories suggesting that COVID-19 does not have a natural origin*"[456] was championed and edited by **Dr. Daszak** ("*Please note that this statement will not have EcoHealth Alliance logo on it…*").[457,458,459] In a linked email conversation between Dr. Daszak and Dr. Ralph Baric and others, he states, "*I spoke with Linfa last night about the statement we sent round. He thinks, and I agree with him, that you, me and him should not sign this statement, so it has some distance from us and therefore doesn't work in a counterproductive way*." "*We'll then put it out in a way that doesn't link it back to our collaboration so we maximize an independent voice*."[460] The February 2020 Lancet statement declared the authors had "no competing interest," but after public concerns about Dr. Daszak's connection with Wuhan-IV gain-of-function research, "*The Lancet invited the 27 authors of the letter to re-evaluate their competing interests*."[461] This Lancet **investigation was terminated** because numerous signatories had been associated with the Wuhan-IV.[462,463]

In another revelation about Dr. Daszak's gain-of-function research cover-up there is also an FOI email that reveals **Dr. Daszak edited a letter** sent by the Presidents of the U.S. National Academies of Sciences, Engineering, and Medicine **to the White House Office** of Science and Technology Policy regarding the origins of COVID-19, including a line stating, "*The initial views of the experts is that the available genomic data are consistent with natural evolution and that there is currently no evidence that the virus was engineered to spread more quickly among humans*."[464] Dr. Daszak also sent Dr. Anthony Fauci an email at the pandemic's outset thanking him for publicly dismissing claims of a COVID-19 lab origin (pg.1150).[465]

While none of this proves EcoHealth Alliance or associates created the precursor for SARS-CoV-2, it does **show that Dr. Daszak actively tried to cover up** his and others' role in gain-of-function research aimed at making bat coronaviruses infective to humans without a zoonosis. Dr. Daszak's collaborative research was largely funded by the **US Military and Government Agencies** (section 2.4.1).

In addition to the various origin cover-ups, the COVID-19 origin narrative has also **sequentially transitioned** as it was **publicly falsified**. This evolving-censoring media narrative originally claimed a

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natural origin even though there was zero evidence for a Wuhan Huanan market zoonosis while covering up a potential gain-of-function origin (i.e., *labeled as a conspiracy by Dr. Daszak et al.*). The narrative then changed to one asserting an accidental release from the Wuhan-IV because the outbreak was first officially diagnosed in Wuhan. However, "if" RaTG13 was the closest genetic relative to SARS-CoV-2 that Wuhan-IV had researched (i.e., 96.2% homology), then an accidental release into Wuhan cannot explain SARS-CoV-2's origin. After all, it needs to be explained in molecular detail how that final 3.8% sequence homology, the high-affinity spike protein-hACE2 binding, and the 2-in-1 double-CGG codon encoding Arginine FCS and nuclear localization signal, among other features, **got added.**

The theoretical specter of another origin beyond Wuhan-IV for SARS-CoV-2 is raised, which could have arisen **inside or outside China** if you considered all possibilities. While allegations of a cover-up by the Chinese Communist Party (CCP) of a potential accidental release in Wuhan should not be ignored or imply guilt (i.e., *HFACR-1:*[466] *pgs.23-37. HFACR-2:*[467] *pgs. 6, 19-29, 58-59, database removal.*[468]), neither should the earlier SARS-like cases among athletes during and after the **Wuhan Military World Games** in October 2019,[469,470,471,472,473,474,475] and the significant increase in Wuhan hospital visits with SARS-like symptoms in the Fall of 2019.[476] The potential importation of SARS-CoV-2 to the Wuhan Military World Games **CONFOUND** a supposed accidental Wuhan Institute of Virology release or Wuhan Huanan market zoonosis, which so far has failed to confirm a progenitor virus or animal host. In assessing a potential China origin, one must also reflect on the lack of published science showing China's science had the proven capability or research **intent** to add an FCS and NLS, LFA-1, etc., and get a functional virus.

Why didn't the **WHO**[477,478,479] and the United Nations Office for Disarmament Affairs (**UNODA**),[480] among others, **more broadly investigate** the SARS-CoV-2 pandemic origin **from the outset** (i.e., all potentialities)? With **confounding** SARS-like cases emerging at the Wuhan Military World Games, a non-China origin was a possibility. Why was the Pentagon-NIH-UNAID-funded Dr. Daszak permitted to be America's sole representative on the WHO 11-member COVID-19 origin investigation team sent to China **one year late**?[481] Considering project DEFUSE's Dr. Daszak (i.e., FCS-intentioned) was caught trying to cover up a SARS-CoV-2 (i.e., FCS-containing) gain-of-function origin with The Lancet statement and associated FOI emails disclosures, while he was one of the WHO group of experts collaborating on COVID-19 vaccine development (i.e., *updated 16/04/2020*),[482] one has to ask if any **conflict-of-interest** was created by Dr. Daszak's inclusion in the belated WHO China origin investigation team (November 2020)?[483]

By applying the **investigatory principle** of investigating those **one degree removed** from Dr. Daszak for his cover-up failure, one would be forced to investigate the Pentagon/DoD and the NIH/NIAID for funding this dangerous research and on/off-shore DoD Biolabs (i.e., *section 2.4 and the Fort Detrick closure in August 2019 for safety violations*[484]), US and other Universities conducting coronavirus gain-of-function

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research (i.e., University of North Carolina at Chapel Hill, among others), Wuhan-IV and China research affiliates and their funders (i.e., including funded international collaborations), Metabiota (section 2.4), and the WHO (incl. its partnership with the DoD in the Ukraine Biological Threat Reduction Program, section 2.4). Yet **China got blamed** even though confounding circumstances and other potentialities existed.

2.4 Was Pentagon-Operated Biolabs Along Russia's Borders Implicated in SARS-CoV-2's Gain-of-Function Origin? Was China Framed & Blamed?

When I reviewed the following 75 cited references and reflected on the issues above, it provoked a question. Could the Pentagon's Biolab/bioweapons research conducted in the USA and countries along Russia's borders, including Ukraine, and in countries between Russia, China, and Iran, and Pentagon-funded gain-of-function and zoonosis experts, somehow (i.e., deep state-sanctioned), be relevant to an alternative SARS-CoV-2 origin? In other words, **was China framed (2014-2017) and blamed (2019)** for COVID-19?

Under the pretext of the Cooperative Threat Reduction program and its subsidiary Biological Threat Reduction Program (BTRP) implemented by the Defense Threat Reduction Agency (DTRA),[485,486,487] the US Department of Defense (DoD) partnered with the Ukrainian Ministry of Health to support the biological detection and reduction of threats posed by pathogens and bioterrorists. In 2005, under this pretext, Senators **Barack Obama** and Dick Lugar entered a partnership with the Ukrainian Government and authorized the construction of a level-3 Biolab in Odesa for processing and researching dangerous pathogens.[488,489,490] The associated BTRP contract effectively gave the DoD full operational control over what Ukraine could do with its deadly pathogens while providing strict confidentiality protections for the USA.[491,492] As of March 2022 the BTRP Ukraine partners also **included** the WHO, the World Organization for Animal Health, and the US Centers for Disease Control and Prevention, among other institutions.[493] Ukraine must have been **strategically important** to have garnered this level of UN affiliate and US domestic agency involvement.

Under this BTRP pretext, the Pentagon operated an extensive network of Biolabs in Ukraine and in other countries along Russia's borders (i.e., Georgia, Kazakhstan, Tajikistan, Kyrgyzstan, Armenia.[494,495]), with Black & Veatch Special Projects Corporation ("Black & Veatch") a key contractor, among others.[496] In 2008 the DTRA awarded one of its Biological Threat Reduction Integrating Contracts to Black & Veatch, including its first task order with Ukraine authorities.[497] Since then it was awarded $337.5 million DoD/DTRA contracts to build and operate Biolabs in Ukraine ($140.2 million,[498] $116.6 million[499]), Cameroon, Iraq, and Armenia, among other countries.[500,501] The DoD/DTRA and Russian Defense Ministry indicate the US invested $200 million in supporting 46 Biolabs in Ukraine.[502,503]

In February 2022 Russian officials claimed the US DoD and Ukraine violated Article 1 of the Biological Weapons Convention by conducting research on highly dangerous pathogens in Ukraine using **gain-of-**

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function synthetic biology technology including **coronaviruses, influenza,** and **filoviruses** (i.e., highly lethal African hemorrhagic fevers).[504,505,506,507,508] Russian officials claimed these programs also assessed virus spread using migratory birds and bats as vectors or intermediate hosts. Ukraine has also faced numerous **mysterious outbreaks** of highly pathogenic diseases in recent years.[509,510,511,512] The Russian Defense Ministry also reported a **100-fold increase** in rare African hemorrhagic viruses, Crimean-Congo hemorrhagic fever, and African Swine Fever in Donbas.[513] How did that **African connection** happen?

On 25/10/2022, Russia filed an official complaint Under **Article VI of the Biological and Toxin Convention** alleging the US and Ukraine participated in banned biological activities in Ukraine. During Russia's **special military operation** in Ukraine, in part motivated by these **aggression-intentioned Biolaboratories**, it obtained "*a variety of documents and evidence that shed light on the true nature of military biological activities of the U.S. and Ukraine on the Ukrainian territory*" and *American and Ukrainian non-compliance with the provisions* of the biological weapons and toxin convention.[514] Why has it taken the United Nations (UN) so long to listen to and seriously investigate claims made by senior Russian Government officials about these Pentagon-operated Biolabs in Ukraine?[515] During this intervening period seemingly devoid of serious investigation NATO has provided tens of billions of Fiat currency dollars in military aid to fund its proxy-hybrid war against Russia, which has destabilized global geopolitics, energy supply, the economy and provokes nuclear brinkmanship. Did the **WHO partnership** with the DoD's Biological Threat Reduction Program in Ukraine create any **conflict of interest** for the UN in investigating Russia's claims and their own belated China-origin investigation?

2.4.1 Follow Pentagon Money from EcoHealth Alliance (Gain-of-Function, Cover-up) to Hunter Biden's part-owned Metabiota to Off-Shored Biolabs (Ukraine, Cameroon)

This section also applies the previously discussed investigatory principle of following the money **one degree removed** from the exposed cover-up. There is no accusation intended, simply a motive to appraise people of the broader associated facts that have largely escaped the mainstream media narrative. Dr. Daszak's gain-of-function/zoonosis research collaborations were largely funded by the DoD/DTRA, NIH/NIAID, and USAID PREDICT dollars. According to USASpending.gov, Dr. Daszak was awarded $69 million in funding for bat coronavirus emergence research (i.e., *from 2014*), bat-borne and other zoonotic potential viruses (Henipaviruses), severe hemorrhagic diseases (i.e., *Crimean-Congo hemorrhagic fever and filoviruses, including Ebola and Marburg*), among other zoonotic viruses.[516] Why did the NIH and EcoHealth Alliance fund coronavirus gain-of-function research at the Wuhan-IV? Was this so the US government could skirt its short-lived (i.e., why so short?) **2014-2017 moratorium** on SARS-CoV-1 and MERS gain-of-function research?[517,518] Or was there a more **strategic reason**?

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EcoHealth Alliance's (EHA) Dr. Daszak also has an extensive and long-standing collaborative history with Metabiota's Dr. Wolfe.[519] The USAID's PREDICT project (Emerging Pandemic Threats program, 2009) lists EHA and Metabiota as core implementing partners,[520,521] while its successor the Global Virome Project lists Dr. Daszak and Metabiota's Chief Scientific Officer in its leadership team.[522] Researchers from EHA, Metabiota, and the Wuhan-IV (Dr. Zhengli Shi) collaborated on a study on bat infectious diseases in China.[523] Likewise, EHA and Metabiota collaborated in numerous studies, including global patterns in coronavirus diversity (2017),[524] Africa coronavirus surveillance (2006-2018),[525] China wildlife-zoonosis risk,[526] viral diversity,[527] Henipaviruses,[528] Ebola,[529] Herpes,[530] and Flaviviruses in bats.[531] This collaborative research was variously funded by the USAID PREDICT project, Google.org, the Skoll and Rockefeller Foundations, and the DoD.[532]

Metabiota is a pandemic tracking and response firm that sells pandemic insurance, conducts zoonotic pathogen research, and operates Biolabs in Ukraine, Georgia, and Cameroon, among other countries.[533] In 2014 Metabiota was awarded a $23.9 million contract from DoD/DTRA for unspecified R&D programs and services in Ukraine and Georgia.[534] Metabiota also shared an office with Black & Veatch in Kyiv,[535] and participated alongside the DoD and Black & Veatch in regional biosecurity meetings.[536]

The $23.9 million DoD/DTRA contract in 2014 likely assisted Metabiota's $30 million Series-A investment in 2015,[537] which Rosemont Seneca Technology Partners led (RSTP),[538] and included Google Ventures in the syndicate.[539,540] RSTP was an offshoot of Rosemont Capital, an investment firm founded in 2009 by **Hunter Biden** and Christopher Heinz (stepson of former US Secretary of State John Kerry). Emails retrieved from Hunter Biden's abandoned laptop (i.e., "*the Biden Laptop illuminated previously convoluted webs of the people you see* ***leading the charge for global governance***")[541] implicated him as a key decision-maker in 2014 between Metabiota and the RSTP investment committee while he made an investment pitch to Burisma executive Vadym Pozharskyi about the "**Ukraine Science**" (see next).[542,543] After this funding round, Hunter Biden may have publicly distanced himself from RSTP because his name was removed from RSTP's website in 2015.[544,545] However, Hunter Biden was still connected with RSTP as Fox Business's posted Biden emails showed he still owned shares in RSTP (2017) and was communicating with RSTP executives about RSTP investments in 2016-17.[546] This connected Biden with Metabiota and the Ukraine Science during the DoD Ukraine contract period while he was generously paid as a Burisma board member.

One Biden laptop Ukraine Science-related email stood out as **very strange** coming from a biotech executive (i.e., Metabiota) to Hunter Biden just after Russia annexed Crimea in 2014; "*I've prepared the attached memo, which provides an overview of Metabiota, our engagement in Ukraine, and how we can potentially leverage our team, networks, and concepts to* ***assert Ukraine's cultural and economic independence from Russia*** *and continued integration into Western society* (now fast forward to 2022).[547] How did Metabiota

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propose to achieve that colossal strategic feat? Meanwhile, Hunter Biden (2014-2019) and Devon Archer, both RSTP Directors, were paid millions as Burisma directors as confirmed by a US Senate Committee investigation: **"Hunter Biden, Burisma and Corruption"** (a **must-read**) during a time when Hunter Biden's father was Vice President and the "*public face of the administration's handling of Ukraine*."[548]

Dr. Nathan Wolfe is the founder and chair of Metabiota and is a World Economic Forum (WEF) Young Global Leader,[549] and Metabiota was a WEF Technology Pioneer in 2021 (for *what technology*?).[550] Dr. Wolfe served on the editorial board of EcoHealth since 2004 and was on DARPA's Defense Science Research Council between 2008 and its disbandment.[551] Dr. Wolfe received more than $20 million from various branches of the DoD, NIH, Google.org, the Skoll and National Science Foundations, and the Gates Foundation, among others.[552] Before Metabiota, Dr. Wolfe founded Global Viral and was director of the Global Viral Forecasting Initiative, which received $5.5 million in grants each from Google.org and the Skoll Foundation to detect early evidence of future pandemics in Cameroon, Democratic Republic of Congo, China, Malaysia, Lao PDR, and Madagascar.[553,554] Metabiota implemented $38.5 million in grants and contracts mainly from the DoD/DTRA and Homeland Security across Central Africa,[555,556] including those linked to the 2014 Ebola crisis in Sierra Leone.[557] Metabiota's surveillance role in the Sierra Leone Ebola outbreak was not without **major controversies**.[558,559,560,561,562] In Cameroon, Metabiota researched corona-, monkeypox-, influenza-, and hemorrhagic fever- viruses (i.e., Ebola). Since 2014 **three of these viruses** were associated with public health emergencies of international concern (**PHEIC**).

Given Dr. Daszak's gain-of-function cover-up exposure, his long-standing research collaborations with Metabiota's Dr. Wolf, and their joint long-standing funders the US DoD/DTRA, the NIH, USAID, and others, a question naturally arises. Did the US government- and/or a transnational- deep state entity via the DoD **operate a second gain-of-function R&D axis**, which was capable of creating coronaviruses with enhanced human infectivity and pathogenicity? If so, was this located in Ukraine, Cameroon, or in a nation along Russia's borders and between China and Iran?

2.5 How A Containable SARS-CoV-2 Outbreak Led to A Pandemic

While a specific SARS-CoV-2 gain-of-function originator is difficult to prove without a thorough independent investigation, what is evident along the chain of events from patient-zero-ish to a full-blown pandemic was a containable outbreak was facilitated in the critical early stages in its global spread by two protagonists failing to fulfill their International Health Regulation mandate obligations (IHR[563] i.e., *the code of international regulations for the control of transboundary infectious diseases*).

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2.5.1 International Health Regulation Mandate Failures That Helped Ignite the Pandemic

In the US House Foreign Affairs Committee Report Minority Staff investigation report (HFACR), "The origins of the COVID-19 global pandemic, including alleged roles of the Chinese Communist Party (CCP) and the World Health Organization,"[564] the following is stated: "*It is important to note that in addition to the obligations imparted on Member States, the IHR requires certain actions and behaviors of the WHO. Among other obligations, the WHO is tasked with conducting global public health surveillance and assessment of significant public health events, disseminating public health information to Member States, and determining whether a particular event notified by a Member State under the IHR constitutes a PHEIC* (i.e., public health emergency of international concern)*. In each of these obligations, the* ***WHO failed to fulfill its mandate***."

The HFACR report identified the following **IHR Article breaches** (pgs. 43-47): (1) **Article 9**: *a failure to assess an unofficial Taiwan CDC email concerning SARS-like cases and report this to member states*. (2) **Article 9**: *a failure to assess unofficial warnings from January 4th by Dr. Ho regarding the human-to-human transmission of SARS-like cases in Wuhan* (University of Hong Kong Centre of Infection, a WHO Collaborating Centre, "UHK-WHO-CC"). (3) **Article 10**: the WHO was empowered to demand the CCP respond to reports made by the Taiwan CDC and the UHK-WHO-CC regarding human-to-human transmission and alert other WHO member states if China refused to cooperate. "*The WHO failed to do so*." (4) **Article 11**: mandates that the WHO promptly transmit to all member states public health information it receives under Articles 5 – 10. The WHO allegedly failed to inform member states about the Taiwan CDC and UHK-WHO-CC unofficial warnings. (5) **Article 12**: see next.

Article 12: Determination of a public health emergency of international concern (PHEIC). According to HFACR (pgs.7-15, 43-47), Director-General (DG) **Tedros failed to follow Article 12** in not declaring a PHEIC on 23/01/2020, instead delaying it one week.[565] Relevant Article 12 decision-making information sent to the WHO or publicly reported before 23/02/2020 included: (1) unofficial communications from the Taiwan CDC (email, 31/12/2019[566]) and the UHK-WHO-CC (04/01/2020). (2) A WHO delegation to Wuhan had already confirmed human-to-human transmission.[567] (3) China's National Health Commission had confirmed human-to-human transmission, including in healthcare workers (20/01/2020).[568] However, there is evidence that Chinese officials knew of human-to-human transmission sometime before the official announcement (HFACR pgs.7-15). The first case was publicly reported in mid-November 2019.[569,570] (4) The identification of a novel causative coronavirus and its genetic sequence and similarity with SARS-CoV was known. (5) Ongoing mass international travel of people in China related to the Spring Festival created global dissemination risk. (6) Confirmation of COVID-19 cases in Vietnam (22/01/20),[571] Thailand (13/01/2020), Hong Kong, Japan, South Korea (21/01/20),[572] Taiwan, and the USA.

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By applying the IHR Annex 2 decision instrument as directed in Article 12 to the above information, by reflecting that half the Emergency Committee members had already recommended declaring a PHEIC, and by reflecting that millions of international trips had departed China by mid-January, Director General Tedros had sufficient information to justify declaring a PHEIC by the 23/01/2020. Why didn't DG Tedros declare a PHEIC on or before 23/01/2020?[573]

Furthermore, according to the WHO criteria for historically declaring influenza pandemics (i.e., *human-to-human spread of the virus in two or more countries in a WHO region, plus community-level outbreaks in at least one other country in a different WHO region*),[574] it well exceeded its Phase 6 criteria by the time it declared COVID-19 a pandemic on the 11/03/2020. By this date, SARS-CoV-2 had already spread to 114 countries.[575] Concomitant with this tardy pandemic declaration, the WHO played a pivotal role in the conditional "**payout triggering mechanism**" of the World Bank's Pandemic Emergency Financing Facility Bond.[576,577] If the WHO called a pandemic before the end of June 2020, the Bondholders would forfeit approximately half of the **$425 million bond**.

2.5.2 Did WHO Advice Against Travel Restrictions Facilitate Global Viral Spread?

Declaring a PHEIC on 30/01/2020 expanded the WHO's authority to coordinate a global response by issuing recommendations on travel and trade restrictions to prevent disease spread. **Instead**, four days later, on 04/02/2020 DG Tedros advised the world there was **no need for measures** that "*unnecessarily interfere with international travel and trade.*"[578] One of China's ambassadors attending a WHO Executive Board meeting even. denounced measures by some countries to restrict travel for people boarding from the Hubei province, saying, "*All these measures are seriously against recommendation by the WHO*." One month later, the WHO updated its recommendations for international travel, "*WHO continues to advise against the application of travel or trade restrictions to countries experiencing COVID-19 outbreaks*."[579] This China travel ban failure helps explain why President Trump was reported as saying the "*U.S. will stop funding to the WHO while his administration reviews its role in "mismanaging" the coronavirus*" (i.e., $400 million pa.).[580] This funding threat was eliminated with the **controversial inauguration** of Hunter Biden's father as US President (i.e., 2020 US election fraud and theft).[581,582,583,584]

Disease modeling suggests that had non-pharmaceutical interventions been implemented, including travel restrictions, one, two, or three weeks earlier in China, **cases could have been reduced** by 66%, 86%, and 95%, respectively, while significantly reducing the number of affected geographies.[585] This study would imply that had the WHO declared a PHEIC on or before 23/01/2020, had the IHR Article breaches detailed in section 2.5.1 not occurred, had the WHO not advised against measures that unnecessarily interfered with international travel and trade, and had an earlier disclosure been made about SARS-like cases during and after the Wuhan Military World Games, then a containable outbreak might not have **ignited a pandemic**.

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2.6 Who Controlled the Potential for Perpetual Mass Death by Coronavirus and COVID-19 Vaccination?

What was an important consequence of conducting coronavirus gain-of-function research in the USA, China, or potentially in Pentagon-operated Biolabs situated in countries between Russia, China, Iran, or elsewhere? It gave a **powerful, strategically aggressive** nation, someone(s), group(s), or an organizational entity (i.e., a transnational deep state) operating outside of democratically elected government powers a potential means to commit genocide (**Control of Genocide-potential**).

What does a tardy declaration of a PHEIC, critical IHR Article breaches, and advising the world not to take measures that restrict travel and trade in the critical early stages of the pandemic teach us? It illuminates the possibility that enhanced global disease spread is a hypothetical consequence of **transboundary disease control decision-making**.

What were the important consequences of not recommending prophylactic **Ivermectin** use in the early stages of the pandemic other than in **controlled clinical studies** (WHO,[586] NIH[587])? This leadership eliminated a cheap (i.e., Bangladesh, US$0.60-$1.80 for a 5-day course) and early prophylactic means of ameliorating the infection, disease, and death impact at the national level during COVID-19 pandemic waves,[588,589,590,591,592] like in India,[593] [594,595] and Peru.[596] Ivermectin blocks the spike protein receptor binding domain's (RBD) interaction with the ACE2 receptor.[597,598,599,600] Ivermectin could have provided a medical countermeasure to the ACE2-spike protein-furin interaction and **neutralized** this gain-of-function modified SARS-CoV-2 **without needing** to vaccinate all demographics. Ultimately, this meant governments who followed these WHO guidelines were **left with no alternative** but to vaccinate their population. Thus, we are made aware of how **scientific advisory boards** of globally mandated healthcare organizations can impact national treatment and vaccination guidelines that control rates of severe disease and death during a pandemic.

What was the consequence of rapidly achieving high national vaccination rates? It failed to prevent symptomatic COVID-19 infection as promoted by governments at EUA. Instead, in my opinion, this rapidly established the **predictable life-long-fixed ADE and antigenic imprinting potential** in the human population before it could be **discovered or uncovered** in government surveillance data with the emergence of antigenically distinct strains (i.e., Delta, Omicron). All things considered, it looks like a perpetual global-scale **human culling biosystem** was created with SARS-CoV-2's non-zoonosis emergence. This will see higher infection, disease, and death rates in the vaccinated each winter or with each pandemic wave. Excess mortality will continue to rise from ADE and antigenic imprinting infection-related disease and from vaccine-associated enhanced disease in at-risk populations with comorbidities and sub-clinical disease. This

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excess death and disease will be explained as unattributable to vaccination, death due to preexisting conditions, sudden adult death syndrome, long-COVID, unascertained natural causes, or some other concocted medical or coroner classification. Governments will stop providing surveillance data by vaccination status, and disease and death classifications will change over time. Meanwhile, statisticians will "process" the continually recategorized data in support of political narratives – **the truth R.I.P**.

2.7 A Pandemic Treaty or Other Legal Instrument before a WHO COVID-19 Investigation?

International Health Regulations (IHR, 2005) provide an overarching legal framework that defines countries' responsibilities and rights in handling and reporting of transboundary infectious diseases of public health concern and criteria to determine if an outbreak constitutes a public health emergency of international concern (PHEIC).[601,602] In January 2022, the US proposed a detailed series of amendments to the IHR 2005 rules to provide more defined criteria, terms, and timelines for alerts, notification, and response to emerging outbreaks,[603] which likely reflected the IHR Article breaches detailed section 2.5.

By contrast, and in addition to the amply provisioned IHR 2005 (+/- amendments), WHO is drafting an additional legal instrument(s) supposedly to protect the world from future infectious disease crises, where in 2019, **it failed**.[604,605] This proposed legal instrument or IHA modification came before an investigation regarding the WHO's conduct associated with:

1) Delays in declaring a public health emergency of international concern,
2) IHR Article breaches and travel advice that may have facilitated COVID-19's global dissemination,
3) Global promotion of the high false positive Corman-Drosten PCR protocol that underpinned EUA-related vaccine efficacy claims, Government policies, vaccine mandates, and media fear-mongering,
4) Failure to urgently investigate all potential origins of the COVID-19 pandemic during its China origins investigation (including in Ukraine and other strategically located Pentagon Biolabs),
5) Recommendation only to use Ivermectin in controlled clinical studies, leaving WHO member nations no option but to deploy predictably harmful vaccines,
6) Global promotion of COVID-19 vaccines that were predictably associated with antibody-dependent enhancement of viral infection and vaccine-associated enhanced disease,
7) Inclusion of Pentagon/NIH-funded coronavirus gain-of-function expert Dr. Peter Daszak in its group of experts collaborating on COVID-19 vaccine development and in its belated China origins investigation.

The World Council for Health (WCH), a coalition of scientists, doctors, lawyers, and civil society advocacy organizations, oppose WHO moves to implement a global pandemic treaty or other legal instruments. According to WCH and other groups, this will increase the WHO's powers over and above IHR (2005) to

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potentially declare unjustified PHEICs or pandemics (i.e., **monkeypox**) and override democratically elected Governments' pandemic disease control strategies with a one strategy-fits all, putting this power into the hands of someone who is not a medical doctor (i.e., DG Tedros).[606] A pandemic treaty could also be used to impose lockdowns and enforce mandatory whole-population vaccination with improperly tested and unsafe vaccines against peoples' free will. WHO could also impose standardized medical care that biases WHO corporate partners' potentially unsafe, ineffective, and expensive treatments over repurposed safe generic drugs and infection-derived natural immunity. Such a treaty would most likely ensure global biosurveillance is implemented (i.e., digital identities, vaccine passports), which could then be linked to government-controlled digital currencies and the potential abuse of power (i.e., freezing your cash).[607,608,609]

Lest we forget, upon digging deeper one can discover other instances of **WHO-associated leadership** that are in my view **uncommon knowledge** (i.e., WHO delays in calling a PHEIC for the *Ebola epidemic,*[610,611] *the genocidewatch.com Open Letter to DG Tedros in 2017 regarding WHO's handling of Sudan's Cholera epidemic,*[612] *an alleged genocide by subordinates of Ethiopia's Tigrayan Peoples Liberation Front Executive Committee, which Tedros Ghebreyesus was an executive member of before his WHO Director-General role,*[613,614] *and WHO's multi-decade R&D initiative for population control by vaccination and its alleged unauthorized testing of a fertility vaccine disguised as a tetanus vaccine in young Kenyan women*[615]).

Why is **New Zealand relying on the WHO** to advocate transboundary disease control and vaccine strategies that fail to ensure and safeguard our national public safety?

3 RESUME & EXPERIENCE

3.1 An Uncommon Vaccine R&D and Risk Factor Experience with Zoonotic-Mutating RNA Viruses

A review of my LinkedIn and ORCID profiles highlights a highly relevant vaccine innovation career. I have uncommon career experience derived expertise in having co-innovated vaccine solutions for zoonotic-mutating RNA viruses that cause respiratory pandemics and used vaccines for more than 36 years. This involved the leadership of company R&D leaders and a global value chain of contract manufacturers and research organizations and expert service providers-partners. This leadership ensured expert regulatory resources and development expertise were provided from day one for all lead optimization, pre-clinical, clinical R&D, and manufacturing process development focused on UK, US, EU, Australia, and China regulatory jurisdictions. Under this leadership, we successfully developed a scalable synthetic universal pandemic influenza-A vaccine to early human proof of concept. We also developed the capability to

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conduct a human influenza challenge study that ultimately broke a global monopoly for such studies.

I am pro-vaccination as a veterinarian and was similarly in the human field until SARS-CoV-2. I have been a global advocate for prepandemic influenza immunization using synthetic universal Tcell vaccines (since 2005), and in combination with Seqirus, GSK, and Sanofi adjuvanted purified and recombinant subunit vaccines (i.e., *hemagglutinin stalk antibody strategies,* since 2008) against influenza-A pandemic threats. Vaccines have permeated the lion-share of my 36-year career (since 1986). I co-championed and co-innovated the company's concept of universal Tcell vaccines and immunotherapies (i.e., *one vaccine for all virus strains and HLA sub-types/ethnicities, mitigating antigenic imprinting*) with benefits (i.e., *synthetic, scalable, stable, ex-cold chain*) for zoonotic-mutation-potential RNA viruses deployable before or just after an outbreak of international concern (since 2002). I raised £23 million from EU corporate pharmaceutical and life science investors for this concept and vision and built and directed a vaccine company (2003-2012).

However, I am not pro-vaccination for mutation-prone coronaviruses using spike protein antigens (since 2004) given their 30-year legacy of antibody-dependent enhancement of virus infection (ADE) and vaccine-associated enhanced disease (VAED). I am against vaccination using genetically modified spike protein antigens that bind to critical physiological receptors lining blood vessels and vital organs (i.e., *heart, lungs, brain, kidney*, gonads, and endocrine) knowing these would cause pathologies with 100% certainty (since 2004, SARS). I believe in the right of choice between the use of superior natural infection-derived immunity over improperly tested and hastily approved harmful vaccination for a disease no worse than influenza in sub-70yr demographics, and for which we already had effective treatments. I am anti-Blitzkrieg speed vaccination campaigns done before predictable ADE could be discovered/uncovered in the surveillance data. I have had long-standing concerns about reverse transcription and genome incorporation, cancer, and autoimmunity for any nucleic acid-based vaccine technology (i.e., any RNA or DNA vaccine, since 2002).

I also have rare research expertise in risk factors associated with zoonotic mutation-prone RNA viruses that cause respiratory pandemics (influenza) linked to environmental-induced immunosuppression, directly and indirectly, consequent to solar-/geo-magnetism (i.e., *circadian system dysregulation-, cosmic ray-induced ionization-, and climate change related cold-stress- induced immunosuppression*) (**hyperlinked preprint study)** (since 2015).

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